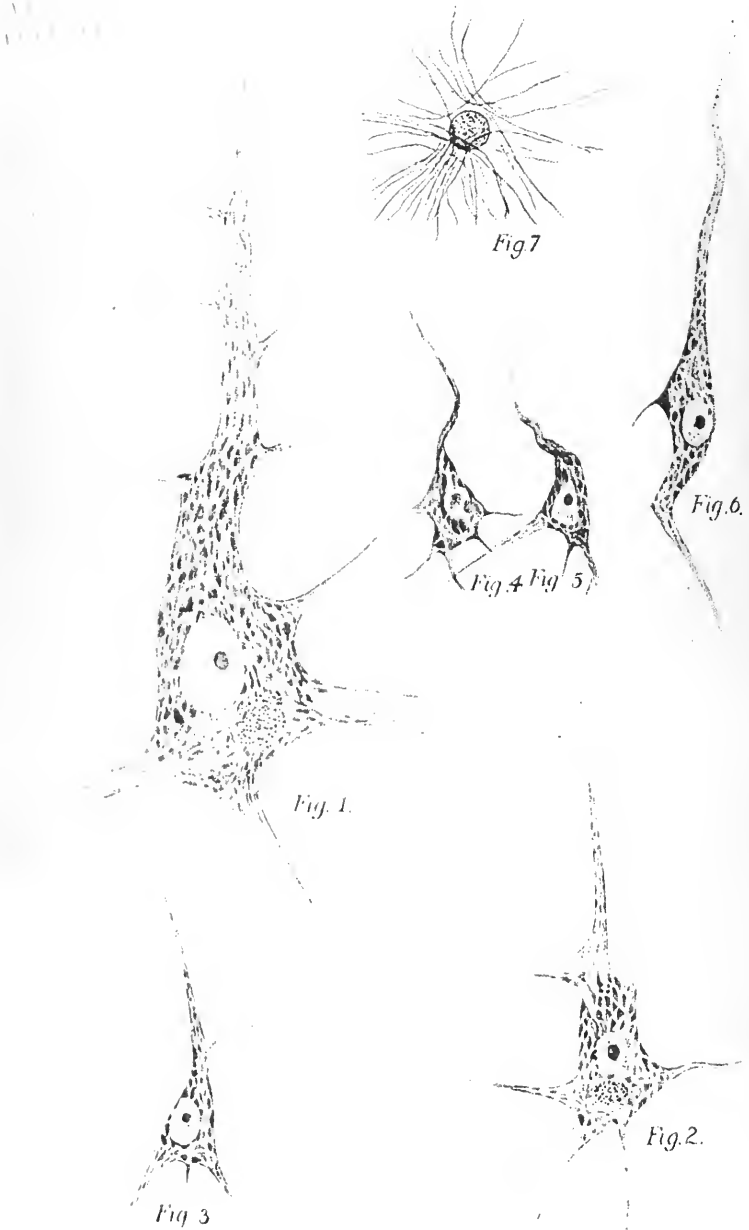

FIG. 1. GIANT CELL, ANTERIOR CENTRAL.
FIG. 2. PYRAMIDAL CELL, 3D LAYER INFERIOR PARIETAL
FIGS 3, 4, 5. SMALL PYRAMIDAL CELLS, 2D LAYER.
FIG. 6. SPINDLE CELL, 4TH LAYER
FIG 7. NEUROGLIA CELL.

TEXT-BOOK

OF

NERVOUS DISEASES

BEING A COMPENDIUM

FOR THE USE OF

Students and Practitioners of Medicine

BY

CHARLES L. DANA, A.M., M.D.

Professor of Nervous Diseases in Cornell University Medical College; Visiting Physician to Bellevue Hospital; Neurologist to the Montefiore Hospital; ex-President of the American Neurological Association; Corresponding Member of the Société de Neurologie, etc.

Fifth Edition

WITH TWO HUNDRED AND FORTY-FOUR ILLUSTRATIONS

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PREFACE.

It is the object of the author in this treatise to present the science of neurology in a concise yet as far as possible complete form. Each subject has been taken, all the available facts regarding it have been ascertained, the writer's own experience has been collated, and with the data thus gathered the chapters have been written. The labor involved in such a task has been very great, but I am encouraged to believe that the result will be a useful one; for the work does not compare or compete with the large treatises which are already in the field nor with the smaller introductory textbooks. I have tried to furnish a book which will be suitable for the student and practitioner and not valueless to the specialist.

The extreme importance of a knowledge of anatomy has led me to pay especial attention to furnishing in a condensed form the most recent accessions to our knowledge of this subject. Starting with the facts that can be gained in ordinary anatomical works, the student can, I believe, acquire a good idea of modern neuro-anatomy with the help of the anatomical chapters given here.

In the classification of nervous diseases and the description of their pathology, I have tried to apply the modern knowledge of general pathology as modified by bacteriology. This I have done conservatively, yet not less than in my opinion is absolutely demanded. A good deal of havoc will be wrought eventually in our conception of the nature of nervous diseases by the newer pathological doctrines; I have made as little change as was consonant with undeniable facts.

The limits placed upon me have made it impossible to furnish a bibliography or to give due credit to every original investigator. Full references to literature are to be found in the works of Hirt, Erb, Seeligmüller, Ross, and Gowers.

In many topics I have been much helped by valuable mono-

graphs of my American colleagues. While a part of these are credited to their proper source in the text, I feel that I ought to refer here to some of the articles that have been of special service to me. They include monographs on Spinal and Brain Tumors by Mills and Lloyd; on Cerebral Palsies of Children by B. Sachs; on Muscular Dystrophies and Writer's Cramp, by G. W. Jacoby and by M. Lewis; on Aphasia, Cerebral and Spinal Localizations by M. Allen Starr; on Cranial Temperatures and on Neurasthenia by L. C. Gray; on Degenerative Neuritis, by W. H. Leszynsky; on Poliomyelitis, by Wharton Sinkler; on Craniometry and Cranial Deformities, by F. Peterson and by E. D. Fisher; on Angioneurotic Œdema, by Jos. Collins; on Brain Tumors, by P. C. Knapp, and on Sclerosis of the Cord, by J. J. Putnam. I am indebted to Tourette's recent treatise on hysteria, to that of Féré on epilepsy, and to the annual volumes of Bourneville on these subjects. The masterly lectures of Charcot and the treatises of Ross, Gowers, Hammond, Hamilton, and Putzel have necessarily been freely used. In the anatomical part I have used the works of Edinger, to whose courtesy I am particularly indebted, the treatise of Obersteiner, and many monographs by Golgi, Marchi, Cajal, His, Waldeyer, and others. My own work in teaching anatomy and pathology has enabled me to do more than present a compilation.

I must finally express my thanks to my publisher, Mr. W. H. S. Wood, for his patience and helpful generosity in my efforts to make my work a production that would be creditable to American neurology.

To the Student.

As a special text-book the present work will be used by two classes of readers, one consisting of those who simply consult it for reference in connection with their cases, the other composed of students who desire to ground themselves systematically in a knowledge of neurology. To this latter class I venture some advice as to the method they should pursue. Neurology is a difficult branch of medicine to master, nor is there any royal road to it. Still, it can be made comparatively easy if its study is undertaken in a proper and systematic way.

In using the present work, the student should first refresh his

general knowledge of nervous anatomy as furnished in ordinary text-books. He should then go carefully over the anatomical descriptions here given of the general structure of the nervous system and of that of the nerves, spinal cord, and brain. A thorough knowledge of anatomy and physiology makes clinical neurology comparatively easy, and in fact reduces much of it simply to a matter of logical deduction.

The student should next master the general facts of nervous pathology, symptomatology, and etiology, for he will find common laws underlying apparently the most varying phenomena. Finally, he must begin to study the special diseases. The number of these is very great; in the present work I have described 176. Many of these are rare, and it would be wrong for the student to burden his memory with the details about them. He need know only of their existence and general physiognomy. There are, however, according to my enumeration, about 65 nervous diseases which are either very common or extremely important, and it is these that the student should master and make part of his working knowledge. Since the distribution and names of the common and rare diseases may be a useful guide, I append here a table and a list:

	Peripheral.	Spinal Cord.	Brain.	Functional.	Totals.
Common and important nervous diseases.....	31	13	12	10	65
Rare.....	56	27	16	11	111
	87	40	28	21	176

The common or important nervous diseases are:

General.—Neuritis, multiple neuritis, degeneration, neuralgia, paræsthesia (5).

Cranial Nerves.—Anosmia, optic neuritis, optic atrophy, ptosis, ophthalmoplegia, abducens palsy, headache, migraine, trigeminal neuralgia, facial spasm, facial palsy, tinnitus, vertigo, ageusia, wryneck (16).

Spinal Nerves.—Cervical neuralgia, hiccough, brachial palsies, single and combined, brachial neuralgia, intercostal neuralgia, herpes zoster, lumbar neuralgia, sciatica, leg palsies (10).

Spinal Cord.—Spina bifida, hemorrhage, pachymeningitis, lep-

tomeningitis, poliomyelitis, transverse myelitis, acute and chronic, secondary degenerations, locomotor ataxia, the progressive muscular atrophies, bulbar palsy, muscular dystrophies, spinal irritation (13).

Brain.—Malformations, hyperæmia, pachymeningitis, leptomeningitis, simple, tuberculous, and epidemic, abscess, hemorrhage, embolism, thrombosis, children's palsies, syphilis (12).

Functional.—Epilepsy, hysteria, the tics, chorea, tetanus, neurasthenia, spermatorrhœa, exophthalmic goitre, occupation neuroses, paralysis agitans (10).

PREFACE TO THE FIFTH EDITION.

The present edition of this work contains some minor changes in regard to the microscopical anatomy of the nervous system and a short chapter, with illustrations, upon the diagnosis of diseases of the cauda equina. I have rewritten the chapter upon myelitis, and have added a chapter on general paresis, since that is a disorder which belongs fully as much to neurology as to psychiatry. Finally, a number of new cuts have been added, some of the old ones have been replaced, and a number of minor corrections in the general text have been made. The elisions have been sufficient to enable me to make these various additions without enlarging the size of the book. I am much indebted to my friend, Dr. George L. Walton, for valuable criticisms and suggestions.

New York, *September 27th, 1901.*

PREFACE TO THE FOURTH EDITION.

Five years have passed since the first edition of this work, and I find it requires a good many changes in order to meet the demands of neurology as it is to-day. I have therefore given the book a very complete revision and have entirely rewritten many parts of it. The chapters on the Microscopic Anatomy of the Nervous System and on the Anatomy of the Spinal Cord and Brain are practically new, and have been made to conform more completely with present views regarding the importance of the neuron. The chapters on the Peripheral Nervous System have been rewritten and rearranged. The articles on Acute Encephalitis, Multiple Sclerosis, and Combined Sclerosis are also practically new, as is also the chapter on Neurasthenia. I have added a chapter on Alcoholic Meningitis. The practical side of our art has been borne in mind; such additions have been made to therapeutics as my experience justified me in recommending. Many of the old cuts have been removed and a large number of new ones substituted. With all this, however, I have not materially added to the bulk of the volume. It has been my purpose from the beginning to have my work fill the space that should be occupied by a compendium rather than that of an encyclopædia.

The number of nervous diseases has not lessened as the years have rolled by, but their grouping and relations are better understood and the essential unity of many groups has been made more distinct. I must still give practically the advice to the medical student which is found in the preface to my first edition. Those who read the present volume will, perhaps, be somewhat disturbed at first by the new nomenclature which has so much to say about neurons, dendrites, and neuraxons. These things, however, have come to stay with us anatomically, and the modern student must become familiar with them. I have to confess, however, that our conception of the neuron has aided us more in our anatomical work

than in our pathology. The practical application of the neuron to disease has not yet furnished us very much help.

I must express my appreciation of the liberal way in which my publishers have co-operated in the difficult task of reconstructing this work. I cannot, also, help expressing the indebtedness which I am under to numerous masters of neuro-pathology and anatomy, whom one must consult and from whom one must learn so much of what he has to present in a systematic treatise like this.

New York, Sept. 22d, 1897.

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DISEASES OF THE NERVOUS SYSTEM.

PART I.

CHAPTER I.

GENERAL ANATOMY, PHYSIOLOGY, AND CHEMISTRY.

In studying the phenomena of life in the human body, we as physicians first learn about its normal structure and functions. We then note the new phenomena which develop when disease comes on, the causes which produce them, and the anatomical changes lying back of them; we group our facts and give the disease a name. Lastly we apply the methods by which the disorder can be expelled and future attacks prevented. In fine, we investigate our subject just as we do that of any branch of natural history. Our study divides itself, therefore, into

Normal anatomy and physiology.

Etiology, a study of the causes.

Symptomatology, a study of the morbid phenomena.

Pathology, under which we include a study of the morbid anatomy and physiology.

Diagnosis, or the method of recognizing and separating out the different groups of diseases.

Prognosis, a forecast of the future course of the malady.

Treatment and prophylaxis.

Again, although nervous diseases show many phases and have many different morbid changes behind them, there are certain features common to all. It simplifies their study, therefore, to learn first what these general features are, just as a person can better survey and plot out country in detail if he knows certain general facts about its boundaries and topography. Hence I shall first present in a general way an outline of the fundamental facts that touch more or less on all forms of nervous disease. My first chapters will

be devoted to a general description of the anatomy and physiology, and then of the etiology, symptomatology, pathology, diagnosis, prognosis, and treatment.

GENERAL ANATOMY.

The nervous system is derived from the epiblastic layer of the developing ovum, and its constituents are modifications of epithelial cells. These cells in the embryo are of two kinds: neuroblasts, which develop into nerve cells and fibres; and spongioblasts, which develop into a supporting structure called neuroglia (His).

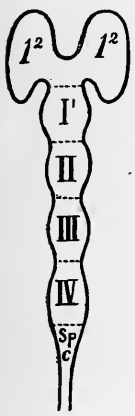


Fig. 1.

The nervous system is composed of:

(*a*) Neurons, which form the nervous tissue proper, and are made up of nerve cells, of their processes, one of which becomes a nerve fibre; and neuroglia.

(*b*) Non-nervous tissue, consisting of connective tissue, blood-vessels, lymphatics, and epithelium.

These tissues are united together to form a central nervous system, consisting of the brain, spinal cord, and the peripheral nervous system. This latter is composed of nerve fibres, and structures attached to the terminations of the nerves, called end-organs, and finally the ganglionic or sympathetic nervous system.

The Arrangement of the Nervous System.—The subdivisions of these parts, and their descriptions in detail, belong to general anatomy. But there have been so many special subdivisions, and particular names given to them in recent years, that I deem it

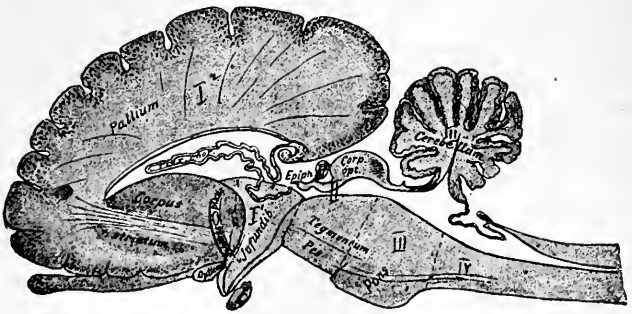


Fig. 2.

necessary, in order to prevent confusion, to describe briefly the subdivisions accepted by modern anatomists. The names here used

are those adopted by the committee on anatomical nomenclature of the German Anatomical Society, and they have also been adopted by a large number of writers on neuro-anatomy.

Beginning with the brain, we find that its particular subdivisions are based upon the embryological development of this organ. As will be shown in more detail later, the brain is developed out of three vesicles, known as the *anterior*, *middle*, and *posterior* vesicles (Fig. 1). The most anterior of these vesicles is the *prosencephalon* or *anterior brain;* the middle vesicle becomes the *mesencephalon* or *mid-brain*, and the posterior vesicle develops into the *rhombencephalon* or *posterior brain.*

Brain.	Prosencephalon (anterior brain). I^1 and I^2	1. Telencephalon.
		2. Diencephalon.
	Mesencephalon (middle brain). II	3. Mesencephalon.
	Rhombencephalon (posterior brain). III and IV	4. Isthmus.
		5. Metencephalon,
		6. Myelencephalon.

The anterior vesicle develops two secondary vesicles: the anterior portion of these, including the corpora striata, olfactory lobes, and the cerebral hemispheres, forms the *telencephalon*, I^2 while the hinder portion of this vesicle, which includes the thalamus and mammary bodies, forms the *diencephalon* (I^1). The middle vesicle is the *mesencephalon*, and it includes the corpora quadrigemina and cerebral peduncles (II). The posterior vesicle is divided, from before backward, into three different parts: (1) the isthmus, which includes the superior cerebellar peduncles and valve of Vieussens, and part of the cerebral peduncles; (2) the *metencephalon* or *hind-brain*, which includes the cerebrum and pons Varolii; and (3) the *myelencephalon* or *after-brain*, which includes the medulla oblongata.

These different parts can be understood better by means of the accompanying figure (Fig. 2), which represents in a schematic way the brain of a mammal (Edinger).

They are intimately connected by strands of nerve fibres, and are connected closely also with the next portion of the nervous system, the spinal cord. The brain and spinal cord are spoken of as a cerebro-spinal axis, and this is in close relation with the peripheral nervous system.

This peripheral nervous system is composed of two portions—first, the cerebro-spinal mixed nerves, whose origin, distribution, and relations are comparatively easy to follow; and second, the ganglionic or sympathetic nervous system. This has relations which are not so easily described, and which are as yet not wholly under-

stood. This portion of the nervous system is composed of two sets of ganglia — one the vertebral ganglia, *i.e.*, the chain of ganglionic masses on each side of the vertebral column, and of certain ganglia connected with the cranial nerves; secondly, a very large number of ganglionic masses distributed in the viscera, and known as the peripheral ganglia. The sympathetic nervous system

FIG. 3.—MULTIPOLAR CELL OF FIRST TYPE. Cell of Deiter. The continuous line is the neuraxon (Van Gehuchten).

is made up very largely of nerves from the cerebro-spinal centres. Motor fibres pass out through the anterior roots of the spinal cord, sensory fibres arise from the posterior spinal ganglia, and both pass in part to the peripheral ganglia and the viscera direct, and part to cells in the vertebral ganglia, with which they are connected. The ganglionic system contains also nerve cells of its own, which are, however, in dependence in the main upon influences from the cerebro-spinal centres. Some of these cells send fibres into the spinal

cord, and according to Collins and Onuf the sympathetic ganglia have representations in the spinal gray matter.

The General Histology of the Nervous System.

The nerve cells form the central body of the neuron and are minute objects varying much in size. The largest are .1 mm. ($\frac{1}{250}$ in.) in diameter and are almost visible to the naked eye. The smallest are .7μ ($\frac{1}{3500}$ inch) in diameter; so that the average diameter is rather greater than that of a white blood cell. In shape nerve cells

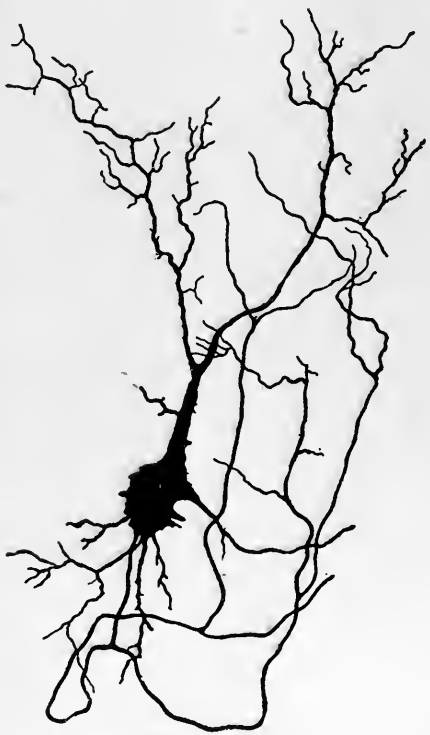


Fig. 4.—Multipolar Cell of Second Type. Cell of Golgi. The neuraxon is given off at the base, soon dividing and subdividing (Van Gehuchten).

are for the most part irregularly spheroidal, but some are pyramidal, others spindle or flask shaped, and others globular. They all give off one or more fine processes or poles, and hence, in accordance with the number of these, the nerve cells are often spoken of as multipolar, bipolar, or unipolar.

In most cells one of the processes is continued on a long way and finally becomes a nerve fibre. This process is called the axis cylin-

der, or *neuraxon* * (Figs. 3 and 4 and plate). The other processes are relatively short and are called protoplasmic processes, or *dendrites.* The nerve cell, then, is a protoplasmic body giving off several dendrites and usually a single neuraxon, the whole forming the neuron. The dendrites branch off irregularly and subdivide, but never anastomose. In some parts of the nervous system they have upon them little nodules or buds, and in the cerebral and cerebellar cortex these are so numerous as to give them the appearance of budded stalks. The dendrites are usually not very long, but in some cells they extend a very great way, reaching many times the diam-

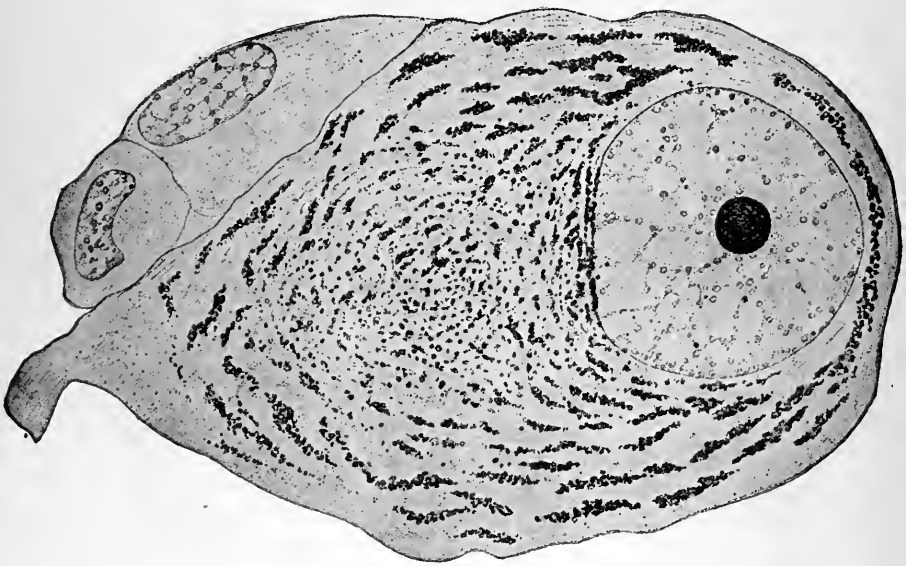


FIG. 5.—NERVE CELL OF POSTERIOR SPINAL GANGLION OF A FROG (greatly magnified), showing nucleus, nucleolus; just to the left of these is the centrosome. The dark masses are the chromophilic granules (Lenhossek).

eter of the cell. The axis-cylinder process, or neuraxon, is given off directly from the body of the cell, as a rule. It very soon becomes clothed with a thin sheath (myelin sheath), and as it passes along gives off branches at right angles, which form what are known as the *collaterals.* The neuraxon and collaterals finally end by splitting up into a number of fine branches, which lose their myelin sheath and form the *end brush* or *terminal arborization.* The axis-cylinder process or neuraxon does not anastomose with other cells either through its own end brush or through the end brushes of its collaterals. The end brushes, however, pass in among the den-

* The name *axon* is also given to it.

drites of other cells, and sometimes closely surround the cell body. In this way one neuron comes into very intimate relation with others, but there is never any true union. *Each neuron of the nervous system is an independent unit.**

There still remain some important facts to be stated regarding the intimate structure of the cell body proper. This is composed of a semifluid albuminous substance, somewhat like the white of an egg, and spoken of in a general way as protoplasm, or, more specifically, as the *cytoplasm*. Within this lies the nucleus and within the nucleus a nucleolus. The body proper is not homogeneous, but is made up of a network of fine fibres or fibrillæ which pass in bundles from dendrite to dendrite and from dendrite to the neuraxon. This fine fibrillary network is not stainable by ordinary dyes and is called the achromatic substance of the cell. Within its meshes and arranged in a rather definite manner are certain stainable bodies called Nissl or chromophilic granules. These form the chromatic substance of the cell. While most nerve cells have these granules, some, such as the granule cells of the cerebellum, do not. The nerve cells which do stain and have the chromophilic granules are called somatochromes, the others are called karyochromes (Nissl). The chromophilic granules are arranged differently in cells of different function. They are believed to represent the nutrient substance of the cell, while the fibrillæ form the conducting and functioning part (Fig. 5 and plate). The cell body usually contains a little pigment (Fig. 6).

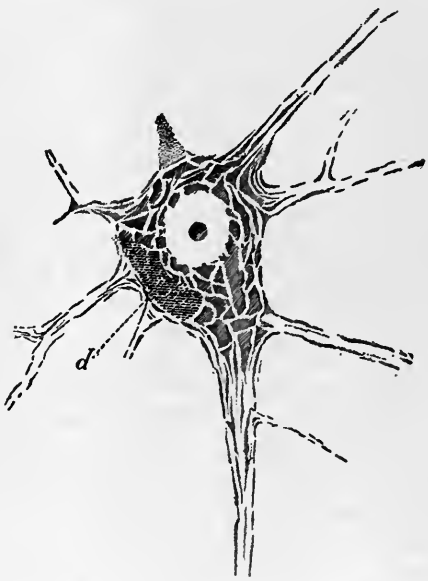


FIG. 6.—MULTIPOLAR CELL. Pigment is seen at *d*.

The nucleus of the cell is a small spherical body, which is also

* This view is now denied by certain authorities, and there is no doubt that in some of the lower animals fibrils from the end-brushes of one neuron pass into the dendrites of another and thence through the body of the cell into its neuraxon. But though there may be this anatomical unity, the genetic and physiological independence of the neurons still continues to be a fact.

made up of a reticulated structure known as the *chromatin network*, because of its taking up dyes and staining very intensely. Besides this network, however, there is a finer network, which is similar to that in the body of the cell, and which is known as the linin network. The chromatin is practically identical with nuclein. Within the nucleus is a smaller body, known as the nucleolus, which stains still more intensely.

Nerve cells are surrounded by a pericellular space, but are not inclosed in capsules, excepting those of the posterior spinal and vertebral ganglia.

Central Nerve Cells.—There are three kinds of central nerve cells, that is, cells lying in the brain and cord, and they are classified in accordance with the peculiarities of the axis cylinders (neuraxons) into:

1. Cells of the first type, or cells of Deiter. These are the kind just described above, and they form the great mass of nerve cells. The neuraxon is continued as a nerve fibre (Fig. 3).

2. Cells of the second type, or cells of Golgi. In these the axis cylinder soon gives off numerous collaterals and quickly splits into a number of fine branches. None of these branches ends in becoming a nerve fibre and none travels far from the cell body (Fig. 4).

3. Cells of the third type, cells of Cajal. In these there are two or more neuraxons. They are found in the superficial layer of the cerebral cortex.

Peripheral Nerve Cells.—The cells of the peripheral nervous system resemble fundamentally the central nerve cells, but undergo some changes in order to adapt themselves to their peculiar functions. Thus the cells of the posterior spinal ganglia have quite peculiar anatomical characters. They are rather large in size, being from 29 to 60μ ($\frac{1}{4\,0\,0}$ to $\frac{1}{8\,0\,0}$ inch). They are spheroidal in shape and have one process, which speedily divides in two, in a T-shaped fashion. They are surrounded by an endothelial sheath, which is analogous to the myelin sheath of the nerve fibre. The cells have one large nucleus with a nucleolus. The body is composed of short granular fibres, which are arranged in a somewhat concentric layer.*

*In the centre of this, in invertebrates and the lower vertebrates at least, is a body which is called a centrosome. This is a minute object which, as a rule, lies outside though near the nucleus, and is surrounded by a radiating area of granules, known as the centrosphere or attraction sphere. It may, however, lie within the nucleus. The centrosome is believed to be the especial organ of cell division, and the dynamic centre of the cell (Fig. 5). The spinal ganglionic cell in the mammal is unipolar; in the lower vertebrates it is bipolar. It has been shown that these two poles have in higher vertebrates simply become fused into one.

The nerve cells of the sympathetic or vertebral ganglia are very like the central nerve cells in the anterior horns. They are multipolar in shape and have dendrites and a neuraxon. The neuraxon goes to other neighboring cells, or it passes on and becomes a fibre of Remak. The cell is surrounded by a connective-tissue capsule lined with endothelium, like those of the posterior spinal ganglia. The peripheral or terminal sympathetic nerve cells lying in the viscera resemble those of the vertebral ganglia. The cells of the special sense organs have many peculiarities of shape and structure,

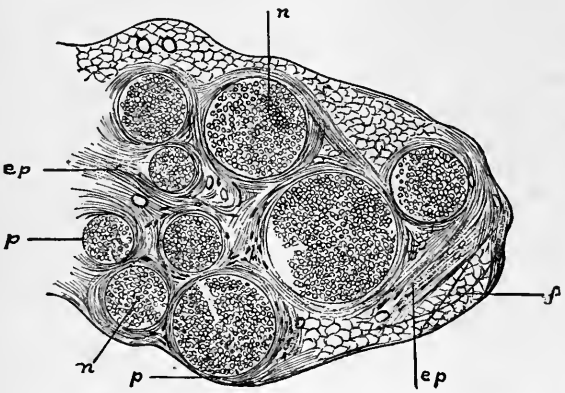


FIG. 7.—FROM A TRANSVERSE SECTION THROUGH THE SCIATIC NERVE. *ep*, Epineurium. *p*, perineurium; *n*, nerve fibres constituting a nerve bundle or fasciculus in cross-section, *f*, fat tissue surrounding the nerve (Klein).

but they are all developments of the same model. In the retina and olfactory bulb there are cells without axis cylinders (spongioblasts of Cajal and granules of olfactory bulb).

Nerve cells are classified in accordance with their shape and number of processes. The multipolar cell is the common type and is found throughout the brain, cord, and sympathetic ganglia. Bipolar cells are found chiefly in the column of Clark of the spinal cord; and unipolar cells in the posterior spinal ganglia. Small nuclear cells and flask-shaped or Purkinje's cells are found in the cerebellum. Besides these there are described in the brain cortex angular, granular, pyramidal, globose, and spindle cells.

THE NERVE FIBRES of the nerve centres are found chiefly in the white tissue or white matter. In the periphery they form the nerve proper of gross anatomy. The peripheral nerve is composed of bundles of nerve fibres called nerve fasciculi (Fig. 7). It is surrounded by a connective-tissue sheath called the sheath of Henle, or epineurium. From this sheath, connective-tissue fibres pass in

and surround the fasciculi. The sheath of the fasciculus is called the perineurium. From the perineurium, strands of connective tissue run in among the ultimate nerve fibres, forming the endo-

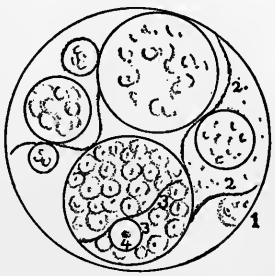


FIG. 8.—DIAGRAM SHOWING THE DIFFERENT PARTS OF THE NERVE. From without inward we have: 1, Nerve with epineurium; 2, nerve fasciculus, with perineurium; 3, nerve fibre and endoneurium; 4, neurilemma, myelin sheath, axis cylinder, primitive fibrillæ.

neurium (Fig. 8). Lymphatic spaces lined with endothelium exist in the layers of the peri- and endo-sheaths. In the nerve centres,

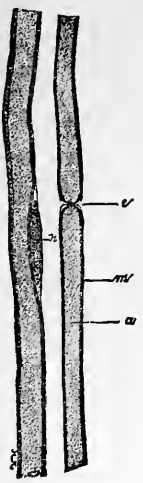


FIG. 9.

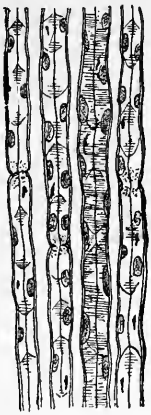


FIG. 10.

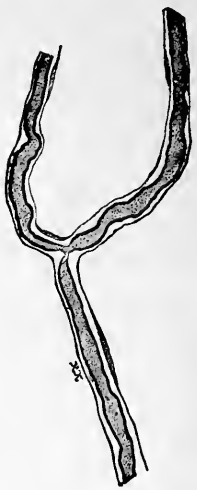


FIG. 11.

FIG. 9.—MEDULLATED NERVE FIBRE. *a*, Axis cylinder; *n*, nucleus; *m*, medullary sheath; *c*, node of Ranvier.

FIG. 10.—MEDULLATED NERVE FIBRE, showing axis cylinder, nuclei of medullary sheath nodes, and oblique incisures of Schmidt.

FIG. 11.—MEDULLATED NERVE FIBRE, showing mode of division.

the nerve fibres have no such sheaths, but are supported by a connective-tissue and neuroglia framework.

The nerve fibre is a long fine strand of tissue varying in diameter.

It may be white or gray, according to its structure. It is composed from within out of (1) an axis cylinder, (2) a myelin sheath, and (3) a neurilemma. (1) The *axis cylinder* is the essential part of the nerve. It is the prolongation of the neuraxon of a nerve cell and consists of protoplasm. It is itself made up of fine fibrillæ (primitive fibrillæ) which run longitudinally. By means of reagents, a transverse striation can be seen also. (2) The *myelin sheath, medullary sheath,* or sheath of Schwann, surrounds the axis cylinder. It is composed of a semifluid, fatty substance, which chemically consists of lecithin, neurin, and some cholesterin. It varies much in thickness, and this is the principal cause of the different sizes of nerves. The myelin sheath is interrupted at regular intervals by constrictions called the "nodes of Ranvier." These constrictions involve the myelin sheath alone. The axis cylinder passes through and the outer sheath (neurilemma) passes over it. There is a little granular matter at the point, called intercellular cement. The nerve fibres, if they divide, always do so at a node (Fig. 11). The part between two nodes is called a nerve segment. In each segment there is an oval nucleus embedded in the myelin sheath. The nodes are about 1 mm. apart.

The myelin sheath is probably developed, like the axis cylinder, from the epiblast, and is closely related nutritionally to the axis cylinder, which it protects and isolates.* (3) The *neurilemma* or *primitive sheath* is a delicate homogeneous covering forming the outermost sheath of the nerve. It is of connective-tissue origin. The sheath is absent in the fibres of the central nervous system and in some fibres of the periphery.

Variations in the Types of Fibres.—In accordance with the arrangement of the sheaths of the nerve fibres, several kinds are described. The principal types are the *medullated* and *non-medullated.*

Medullated nerve fibres make up the bulk of the white matter of the brain and cord and cerebro-spinal nerves. They consist of a myelin sheath and axis cylinder, and may or may not have a neurilemma. Fibres with myelin sheath, but without a neurilemma, make up the white matter of the central nervous system.

Non-medullated fibres, or fibres of Remak, occur principally in the sympathetic system, but they are also found in the cerebro-

* Between it and the axis cylinder Mauthner describes a membranous sheath (axis-cylinder sheath). Another sheath is said to be between it and the neurilemma (medullary sheath). By means of certain reagents, oblique lines (incisures of Schmidt) or a reticular appearance may be developed (network of Gedvelst). These appearances are, perhaps, artificial.

spinal nerves. They are grayish and faintly striated, and consist of axis cylinders, with a thin, homogeneous, nucleated sheath lying directly upon them. This sheath, however, cannot often be demonstrated (Schaefer).

Naked axis cylinders are found in the peripheral terminations of nerves as well as in the brain and cord.

Size.—The nerve fibres are of two kinds as regards size. The

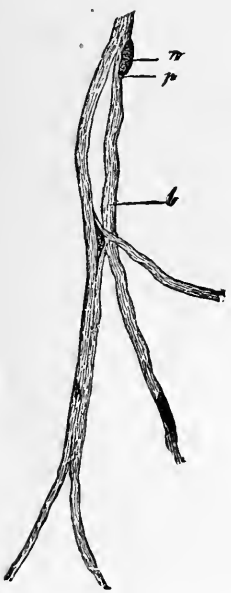


Fig. 12.

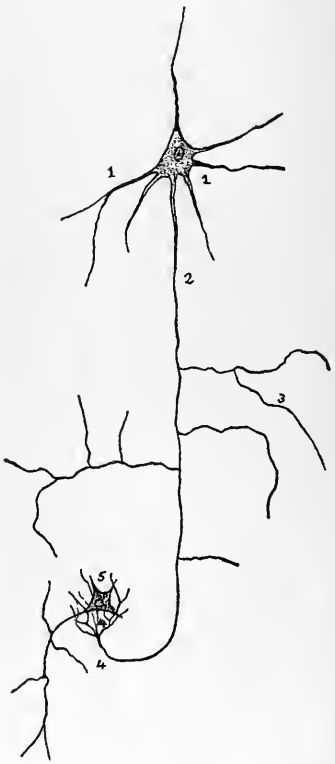


Fig. 13.

Fig. 12.—Non-medullated Nerve Fibre. *n*, Nucleus; *b*, striations.

Fig. 13.—Diagram showing the Neuron and Mode of Connection between Nerve Units throughout the Nervous System. 1, Nerve cell; 2, nervous process; 3, collateral; 4, end brush; 5, nerve cell.

small fibres are about 2μ or $\frac{1}{12000}$ inch in diameter, the large 20μ or $\frac{1}{1200}$ inch. The small fibres are connected with smaller cells, and either run a shorter course or are distributed to the involuntary muscular fibres of the blood-vessels and viscera. The motor fibres are larger than the sensory.

The peripheral nerve fibres, except the optic, have no neuroglia;

they terminate in fine fibrillæ among epithelial cells, or in special end organs.

The *central nervous fibres* make up the white matter of the brain and cord. They are, like the peripheral nerves, the prolongations of the neuraxons. They are composed of an axis-cylinder process and myelin sheath, but have no neurilemma, and probably no nodes. At frequent intervals each fibre gives off branches at right angles forming the "collaterals."

Connections of Nerve Cells and Nerve Fibres.—One nerve cell is never connected directly with another, so far as anatomical investigation can show. One nerve process becomes an axis cylinder, receives a myelin sheath, gives off collaterals, and finally breaks up into a fibrillary "end brush" surrounding a cell, but not passing into it. There is physiological, but no apparent anatomical continuity (Fig. 13).

The Neuroglia.—The supporting tissue of the peripheral nerves is connective tissue only; that of the central nervous system is con-

FIG. 14.—NEUROGLIA CELLS.

nective tissue and, in addition, a peculiar substance called neuroglia. The neuroglia or supporting tissue of the nervous centres is derived from the epiblast. It is composed of cells with very numerous and finely ramified processes, which make a supporting network about the nerve cells and fibres (Fig. 14). The neuroglia cells are some-

times known as "spider cells." They differ somewhat in size and shape, but not in general characteristics. The cell body is composed of granular protoplasm, lying in which is a large nucleus, within which is the nucleolus. The body of the cell is small in amount in proportion to the nucleus. The fibrillary processes form a felt-like network, and in regions where there is much neuroglia tissue this looks like a homogeneous matrix. It is, however, made up of the fine fibrils. These connect with the walls of the blood-vessels.

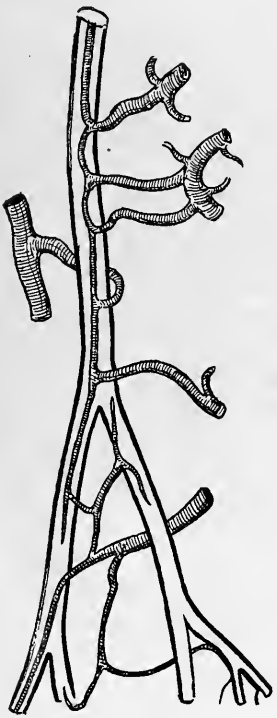


FIG. 15.

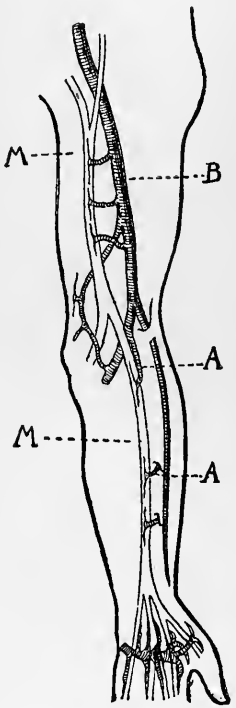


FIG. 16.

FIG. 15.—ARTERIES OF SCIATIC NERVE (QUÉNU AND LEJARS).

FIG. 16.—ARTERIES OF THE MEDIAN NERVE. *AA*, Branches to the nerves; *B*, brachial artery; *M*, median nerve (Quénu and Lejars).

Weigert has shown that the processes become changed in their chemical and physical character, so that they take a different stain from that of the cell body itself, and thus form a really separate structure (Plate I., Fig. 7). In inflammatory conditions the cells multiply, swell up, and assist in carrying off irritating products (scavenger cells of Lewis). Neuroglia tissue is richly deposited about the central canal of the spinal cord, beneath the ependyma of the ventricles, and beneath the pia mater of the brain and cord. The epi-

thelial cells of the central canal and ependyma of the ventricles send down fine processes which form a minor part of the supporting framework.

THE NON-NERVOUS TISSUES—*The Blood-vessels.*—The peripheral nerves are richly supplied with blood. Each nerve receives arterial supply from many different branches, but always from the same general source. The artery passes to the nerve sheath obliquely, then divides dichotomously and sends branches a long distance up and down on the sheath. It may pierce the sheath, however, first, and then divide, as above described. The dichotomous branches send off arterioles and capillaries, which form plexuses about the nerve fascicles. These are "the interfascicular arcades." The arteries subdivide in such a way as to prevent sudden impact of a large blood stream into the tissue of the nerve. In this respect the nerve circulation resembles that of the brain and cord. The veins subdivide dichotomously, like the arteries. They freely anastomose with the muscular veins, so that muscular action helps nerve circulation. The veins of the superficial nerves connect with those of the deep nerves (See Figs. 15, 16, 17).

The blood-vessels of the spinal cord and brain will be described later.

Lymphatic vessels and spaces are found in the epineurium and perineurium. There are no distinct lymphatics in the fasciculi, but lymph spaces probably exist.

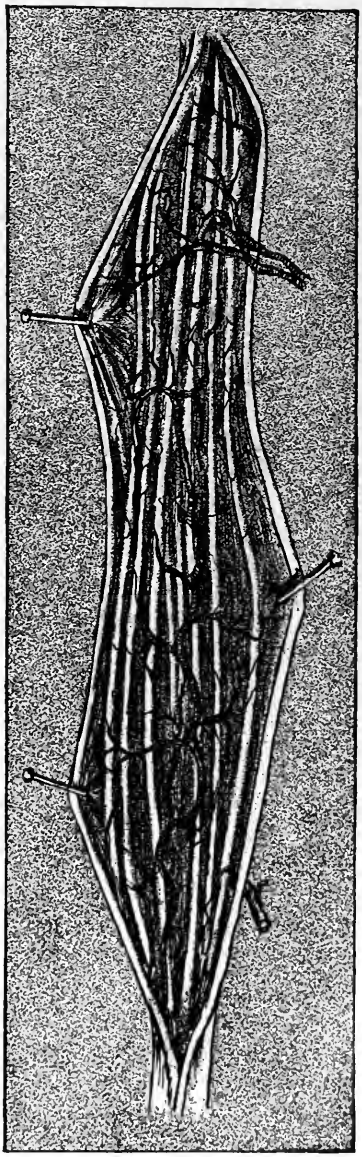


FIG. 17.—INTERFASCICULAR DISTRIBUTION OF ARTERIES (QUÉNU AND LEJARS)

The Neuronic Architecture of the Nervous System.

Having described the component parts, I shall now show the way in which these parts are arranged to form the nervous system.

The nervous system, as already shown, is composed of single nerve units that are called *neurons*, and the neuron is made up of a cell body and numerous processes, one of which is the neuraxon, the others the dendrites.

The neuraxon has always the function of carrying impulses away from the cell; it is a cellufugal fibre; the dendrites, however, bring impulses to the cell, and are cellupetal in function. The dendrites are in contact with end brushes of the neuraxons of other cells, and in this way receive the nerve impulse and transmit it to the cell body. The nervous system is thus a mass of neurons which are packed closely together, and form with each other most intricate relations, but never connect directly one with another. No nerve fibre or dendrite of one cell anastomoses with that of another, as blood-vessels do. Each neuron is absolutely anatomically independent. (See p. 7, note.)

It is the purpose of this neuronic mass to receive impulses from within or without the body, to transfer and modify them, and to send out impulses in such way as properly to control the vital functions and keep the individual in proper harmony with his environment. The nervous system is a great receiving, regulating, controlling, and discharging machine, the machinery being the neurons, the force that works in it being called nervous energy. The nerve cells are the reservoirs and direct generators of this force, while the dendrites and neuraxons receive and distribute it. The nerve cells are massed together for the most part in the brain and spinal cord, forming the gray matter, while the neuraxons as distributors make up the white matter and the cranial, spinal, and sympathetic nerves. It is convenient to make a division therefore into the central nervous system, or cerebro-spinal axis, and the peripheral nervous system, which includes the cranial and spinal nerves and the sympathetic nervous system. Since the nerve fibres of the brain and cord are white in texture, while the cells in mass are of gray color, it is very easy to distinguish the deposits of cells from the fibres and thus make subdivisions of the central nervous tissue. One portion of this gray matter is found deposited in the centre of the spinal cord, extending up to the floor of the medulla, thence underneath and around the aqueduct of Sylvius to the floor of the third ventricle. This is called the central gray matter. An-

other deposit, much larger in amount, covers the whole of the cerebrum and cerebellum, and forms the cerebral and cerebellar cortex. Smaller deposits make up the great basal ganglia, corpus striatum, optic thalamus, and corpora quadrigemina, besides several small deposits (the small basal ganglia), such as Luys' body and the red nucleus.

The peripheral nervous system contains nerve cells, as well as fibres. Their anatomical arrangement is easily understood, but their relation to the central nervous system is less simple and has only recently been made out.

Most of the nerve cells of the periphery, aside from those in the special sense organs, like the eye and nose, have been considered to belong to a kind of special or "sympathetic" nervous system. There is no harm in using this nomenclature, provided it is understood that this system is really a part of the rest of the nervous system and not in any sense an independent mechanism. The peripheral nerve cells of this system are collected in two distinct groups. One is made up of the vertebral and cranial ganglia of the sympathetic, and the second of the peripheral ganglia of the sympathetic, such as the cells of the plexus of Auerbach, Meissner, and the various interstitial ganglia of the glands and muscles.

I come now to a description of the general arrangement of these various nerve units; and here I must suppose that my reader has a knowledge of the ordinary anatomy of the subject.

The nerve cells of the ganglia on the posterior spinal roots furnish the best starting-point in an attempt to trace out the connections. These cells give off a single process, which quickly divides in a T shape. One branch of the T passes peripherally through a mixed spinal nerve to the skin, forming a sensory nerve. The other passes centrally, enters the posterior spinal roots, and breaks up into little filaments, which surround a nerve cell in the posterior horn or analogous nuclei. This forms the first or outer sensory neuron. The outer branch of the spinal ganglion cell which went to the periphery as a sensory nerve was its *dendrite*, or protoplasmic process, which has evolved into a sensory nerve and is *cellupetal* in function. The other process is the neuraxon proper and it is *cellufugal*, carrying impulses away from the ganglion cell into the cord.

The next neuron begins as a cell in the posterior horn, or in like parts. It sends a neuraxon up the spinal cord, a collateral branch passes to the cerebellar cortex, while the direct fibre surrounds a cell in the optic thalamus. This forms the second sensory neuron. The cell in the thalamus gives off a neuraxon which passes to the gray matter of the cerebral cortex, and here it either directly affects

the cells in this region or does it through the medium of another shorter neuron, which is called "associative."

Thus each sensory impulse from the periphery reaches the conscious centres of the brain by passing along three or four neurons. The primary neuron in all cases lies mainly outside the central

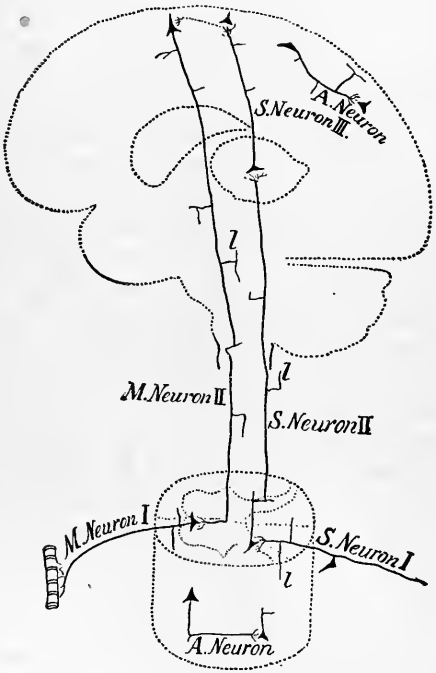


FIG. 18.—DIAGRAM SHOWING THE ARRANGEMENT OF THE NEURONS OR NERVE UNITS IN THE ARCHITECTURE OF THE NERVOUS SYSTEM. *M. Neurons I.* and *II.*, Motor neurons; *S. Neurons I., II., III.*, sensory neurons; *A. Neuron*, associative or commissural neuron.

nervous system and forms a sensory nerve. The sensory nerves do not therefore *arise* in the cord or medulla, but have their terminal nuclei there.

The neurons of the brain cortex cannot yet be distinctly classified, and I shall not attempt it here at all. The matter will be brought out more fully in connection with the anatomy of the brain. It is sufficient to say that nerve units connect together the cerebellum and cerebrum with the basal ganglia, the frontal lobes and the cerebellum, the two hemispheres of the cerebrum, and different areas of the cerebral cortex. Leaving out of consideration these neurons, which are largely psychic in function, we start with the large motor cells in the central convolutions of the brain. These

send down neuraxons, which pass into the spinal cord and surround the cells of the anterior horns. They form the primary or central motor neurons. The anterior-horn cells send off neuraxons, which pass out through the anterior roots and thence to the voluntary muscles. These are the secondary or peripheral motor neurons. Besides this there are groups of cells in the lateral horns and central parts of the spinal cord which send off neuraxons that also pass through the anterior roots, but they leave the cerebro-spinal nerves and enter the vertebral sympathetic ganglia. Here they in part surround the cells of these ganglia and have their terminals there. These sympathetic ganglion cells in turn send neuraxons, which pass in the sympathetic nerves to the peripheral ganglia, where they meet a third group of neurons. They also connect with the other ganglia of their own class and send neuraxons through the posterior spinal roots to the cord. It is not known with what neurons higher up in the nerve centres the lateral-horn cells are connected, but probably with cells in the thalamus.

Such in outline is the neuronic architecture of the nervous system. I do not attempt here to work out the neurons of the special senses, nor to introduce the spinal-cerebellar neuron. This will be done later. It is sufficient to say that the studies in this direction show a marvellous harmony as well as beauty in nature's scheme.

The neuronic architecture is shown in the accompanying diagram (Fig. 18).

GENERAL PHYSIOLOGY.

The Peripheral Neurons.—The nerves which run between nerve centres and end organs carry impulses each way. They are, therefore, divided into the *afferent*, centripetal or in-going, and *efferent*, centrifugal or out-going. The old division into motor and sensory nerves will not answer, for there are many out-going nerves which are not motor. The *afferent* nerves are:

1. The sensory, including:

- Nerves of general sensations.
  - Pain nerves or pathic nerves, heat and cold or thermic nerves.
- Nerves of special sensation.
  - Tactile, including
    - Contact,
    - Pressure,
    - Locality.
  - Nerves of muscular sense.
  - Nerves of special sense f smell, sight, taste, hearing, and space.

2. Excito-reflex nerves.

The *efferent* nerves are:

1. Motor nerves, going to voluntary or striped muscles, heart muscle, smooth muscle, including the vaso-constrictor and dilator nerves.

2. The secretory. These act upon glands. Impulses to the blood-vessels (vasomotor) generally accompany the secretory impulses.

3. The trophic.

4. The inhibitory. These nerves control muscular movements, secretion, perhaps also nutrition.

We must admit that afferent and efferent impulses take place also between end organs and certain (so-called) sympathetic ganglia. In other words, the cerebro-spinal axis is not always the centre. But these subordinate and peripheral centres are normally in connection with the spinal cord and may be influenced by it or by higher parts.

The Central Neurons.—There are intercentral or commissural neurons, which connect different parts of the cerebro-spinal system together. Some of these connect symmetrical parts on each side together. They have co-ordinating function. Others connect higher with lower centres. These latter are made up of ascending and descending fibres. Higher centres send down impulses by the latter, which may stimulate or inhibit lower centres. In the peripheral nervous system we have also *end organs*. These are delicate and in some cases complex arrangements of the nervous and other tissue at the periphery of the nerves. Their object is to allow the nerves to be irritated by special *stimuli* which would not otherwise affect them, *e.g.*, light or sound. Their object is also the proper utilization of efferent impulses upon other tissues. There are end organs, therefore, for both sensory or afferent and for efferent nerves. The end organs of the afferent nerves are—eye, ear, taste buds, corpuscles in the Schneiderian membrane, various tactile cells and bodies, the space-sense organ.

For the efferent nerves—neuro-muscular corpuscles in the voluntary muscles, local ganglia about the arteries, local ganglia in the glands. Trophic end organs are not known. In many cases the end organ is nothing but the terminal fibre of the nerve. This loses both medullary sheath and neurilemma, leaving only the axillary cylinder. It then splits up into a terminal plexus, or else without splitting passes between and around the cells which it is to affect.

The nerves, centres, and end organs thus described may be arranged in mechanisms, each mechanism subserving a special function. These form the *mechanisms of the nervous system proper*, and those of the other organs of the body.

They may be classified somewhat as follows:

Cerebro-spinal.	I. The psychical mechanism. II. The automatic mechanisms or the mechanism of inherited and acquired aptitudes. III. The sensory mechanism. IV. The voluntary motor mechanism. V. The reflex mechanism.
Visceral mechanisms.	The secretory mechanism. The trophic mechanism. The thermic mechanism. The vasomotor mechanism.

Various of the simpler mechanisms are combined to form those more complex. Thus the automatic and psychical mechanisms embrace in their activity other mechanisms of lower grade.

In the same way mechanisms are combined for the regulation of visceral functions. Thus we have the cardiac, respiratory, and other visceral mechanisms.

The Cell-body of the Neuron.—In the working of these mechanisms the nerve-cell body is the agent which generates the energy of all nerve force, by which impulses are started, controlled, and distributed. The larger the nucleus of the cell in proportion to its protoplasmic body, the more stable or less sensitive the cell. The larger the amount of protoplasm relative to the nucleus, the more active the discharging power of the cell. The nucleus is the part of the cell body which is essential to constructive metabolism. By means of it the cell builds up its protoplasmic substance. When the nucleus dies, the cell may live or function for a time, but it lives only on what has been stored up; it can build no more. Nerve cells with few exceptions (spinal ganglia) have no centrosomes; they cannot divide and multiply. Once dead they cannot be restored.

The nerve fibres conduct impulses generated by nerve cells. These impulses travel at the rate of about 100 to 120 feet per second. It is less in visceral nerves (25 to 30 feet per second). There are no electrical currents in normal living nerves (Landois) except when an impulse travels along them. Then an electrical current travels along with the impulse. It is called the current of negative variation. The irritability or excitability of a nerve is the power it has of responding to a stimulus. When a constant electrical current is passed along a nerve its irritability is modified. This modified condition is called *electrotonus*. When a nerve impulse passes up an afferent nerve and is then reflected along an afferent nerve, it is called a *reflex action*. The time required for this process is

called the *reaction time*. This averages from 0.125 to 0.2 of a second.

CHEMISTRY.

The specific gravity of nervous tissue is about 1.036; that of the brain is 1.038; of the spinal cord and nerves, 1.034 (Bischoff, Krause). The reaction is alkaline, but this is lessened by activity, owing to the development chiefly of lactic acid. The gray matter is less alkaline than the white.

The nervous system has the following composition (Baumstark, quoted by Hammarsten):

	White Matter.	Gray Matter.
Water in 1,000 parts	695.35	769.97
Solids	304.65	230.03
Protogon { Cerebrin / Lecethin (neurin) }	25.11	10.08
Insoluble albumin and connective tissue	50.02	60.79
Cholesterin	45.12	23.81
Nuclein	2.94	1.99
Neurokeratin	18.93	10.43
Inorganic salts	5.23	5.62

Water makes up nearly three-fourths of nervous tissue, there being more in the gray than in the white matter and least in the sympathetic nerves. The inorganic salts amount to about .5 per cent. The largest single constituent is phosphorus (Breed) combined with potassium, sodium, magnesium, calcium, and iron, forming phosphate salts. Of other constituents chloride of potassium is the most important.

Protogon is a very complex substance of a fatty character, containing nitrogen and united with glycerin-phosphoric acid instead of glycerin. It is said by some to be made up of two bodies, cerebrin and lecethin, the latter containing an ammonia compound called neurin. Protogon is especially found in the white matter. The gray matter contains nuclein, a very important substance in cell metabolism. Nuclein ($C_{29}H_{49}N_9P_3O_{22}$, Miescher) is composed of nucleic acid, a substance rich in phosphorus and a variable amount of albumin. The gray matter, *i.e.*, the nerve cells, contains also various albuminous substances. The nuclein and the allied substance nucleo-albumin are called albuminoids (Halliburton). They both contain phosphorus and are found chiefly in the nucleus. The albuminous substances, called also proteids by Halliburton, have little or no phosphorus, and make up the most of the cell body or cytoplasm.

CHAPTER II.

THE CAUSES OF NERVOUS DISEASES.

Nervous diseases are produced in part by predisposing influences which may be likened to a fecund soil; in part they are due to exciting causes, which are like the seeds dropped upon the soil in the accidents of life.

Heredity is the most serious and important of these predisposing causes, in particular of those neuroses that are constitutional and are not the results of bodily accidents. A nervous disease, however, is rarely directly inherited. Parents do not pass down special maladies, but only a general tendency to nerve disease, which is not developed into any distinct trouble unless some disturbing cause arises. Nervous parents may have children who have unstable, over-irritable, and inadequate nervous systems. Such persons have what is called a *neuropathic constitution* or diathesis. This diathesis may be transmitted when the parents, though not especially neurotic, suffer from syphilis, alcoholism, and diseases of malnutrition, like tuberculosis. So far as the offices of parentage go, persons of great talent in affairs, or great artistic genius in any direction, may be counted as neurotic and are very likely to have children of neuropathic constitution. This is less apt to be the case when one parent is of stable and lymphatic type. If persons having not simply a nervous constitution but distinct nervous or mental disease marry, their children are liable to serious nervous or mental disease. The intermarriage of blood relations such as first cousins does not lead to neurotic children if the parents are not both of that class, or are of robust health and dissimilar temperaments. Injuries or even severe shock to the mother during the early months of pregnancy sometimes leads to nervousness in the offspring. The mother transmits neuroses more often than the father. There are certain rare nervous diseases which appear in different branches and members of a family, such as an uncle, cousin, nephew, and son. These diseases may pass also by direct inheritance from parent to child, or may skip a generation. They are called "family diseases," and are of the nature of congenital defects, like webbed fingers or clubfoot.

Morbid traits that have become fixed in a family reappear at

about the same age in the descendants. If these traits or tendencies are disappearing from the family, however, they appear later in life with each successive generation in the descendants. If they are becoming more intensified, they develop at an earlier age in the successive descendants. Thus migrainous attacks which have existed in a family usually appear between twelve and fifteen. If now they do not develop till the age of thirty it shows that the migrainous taint is dying out.

When a nervous disease develops in a grandchild, having skipped a generation, it is called a manifestation of atavism. Atavism is a very slight factor in nervous diseases, and rarely goes back more than two generations.

Degeneration is the name given to a condition in which there is a morbid deviation from the normal average. It is almost always an inherited state, and the word degenerate is often used to indicate a person who has a hereditary neuropathic constitution. Degeneracy in a moderate degree often accompanies great mental powers, especially of the artistic kind, and it is almost invariably associated with genius. It is quite compatible with mental soundness and a fair degree of physical health. Those who have unusual mental gifts and degenerate characteristics are called superior degenerates. The criminal and the insane and erratic and eccentric persons of weak judgment have also the neurotic constitution, and are called inferior degenerates. The weak minded, imbecile, and idiots form the lowest class of degenerates, and are called the *debiles*. The degenerate tends to sterility, and if two degenerates marry, and have children, their children are likely to be more abnormal than the parents. Degenerate families tend to die out. But this tendency can be avoided by the infusion of sound blood.

Age.—In infancy and early childhood, nervous diseases are rather frequent on account of the accidents at birth, the liability to infectious fevers, and malnutrition, and the high degree of sensitiveness of the yet immature nervous system. Still, a carefully watched infant is relatively safe. Motor disorders, such as paralyses, convulsions, and chorea, are much the more common troubles. At the time of puberty sensory disorders, such as headache and migraine appear, and often epilepsy, hysteria, and disorders of sleep. Hereditary tendencies to nervous disease also begin to develop at this time or a little later. At the period of adolescence, the maladies already mentioned also may be brought out; but in addition neurasthenic, morbid sexual, hypochondriacal, and insane tendencies are seen. From maturity to the time when degenerative changes begin, forty to forty-five, the individual suffers from those

nervous disorders brought on by accidents, injuries, prostrating attacks of sickness, overstrain, infections, indulgence in alcohol and narcotics, and the abuse of the bodily functions. At and after the climacteric, one sees oftenest such maladies as result from vascular disease, apoplexies, softening, severe forms of neuralgia, and spasm.

Sex.—Sensory and functional disorders are more frequent in women; motor and organic disorders more frequent in men.

Condition and Occupation.—No general facts will be laid down here. Celibates, however, it may be said, suffer more from nervous disorders than married people. It will be shown later that certain occupations entail special nervous disorders and that indoor life promotes functional nervous diseases. The influence of education in the development of nervous diseases is very great, but it can be best considered in connection with special diseases.

Habits.—Excessive indulgence in alcohol is a most prolific cause of nervous disease, chiefly by the action of this substance on the blood-vessels and the stomach. Excesses in eating, in tea-drinking, irregularity in sleeping, and bad habits of working predispose to nervous disease. Sexual excesses are usually the result rather than the cause of nervous disorders. They are the evidence of mental more than of nervous weakness.

Climate and Civilization.—Nervous diseases are most frequent in temperate climates, and in those which are dry and elevated. They increase with the progress of civilization and the greater strain, complexity, and luxury of modern social life. Those organic nervous diseases which are largely dependent on vascular disease are frequent in the poorer classes, among whom syphilis, alcoholism, and bad feeding prevail. Functional and degenerative disorders are frequent in the higher classes. Nervous diseases, if we except those of the degenerative type, prevail more in urban populations.

Diathesis.—The rheumatic and gouty diatheses predispose to nervous troubles, more especially those which are of a peripheral and functional nature. Lithæmia, a condition in which the products of tissue waste are not properly oxidized and eliminated, has a similar influence.

Trauma and Shock.—Exhausting hemorrhages and trauma may be the direct cause of or may predispose to nervous disease. Trauma and shock may cause functional diseases such as neurasthenia, or may lead to the development of insanity or indirectly to degenerative organic disease. Mental shock, and especially a fright, oftener

than severe bodily injury, leads to the development of functional neuroses.

Infections.—In comparison with their frequency, the infective fevers are not great factors in producing nervous disease, but practically they often play an important part. Scarlet fever is the most dangerous disorder in this respect. Measles perhaps ranks next; then follow influenza, diphtheria, typhoid fever, and pertussis. Among chronic infections syphilis ranks first; malaria, the pellagra, and beriberi are also to be mentioned.

Poisons.—Alcohol, tea, coffee, and lead, mercury, copper, and arsenic, are to be placed among the frequent causes of nervous disease. Alcohol in excess is justly credited with exerting the most sinister influence on the nervous system, even leading to an acquired state of degeneration.

Reflex Causes.—Among other causes are local disease of viscera, such as renal, uterine, and ovarian diseases, dyspeptic and liver disorders, visual and auditory troubles. Reflex irritations are distinctively exciting causes, and with few exceptions they cannot cause a nervous disease unless there is a predisposition to it. They may, however, cause many distressing nervous symptoms, such as pain, spasm, and even convulsion.

CHAPTER III.

GENERAL PATHOLOGY.

The nervous system is composed of nerve cells and nerve fibres, forming neurons, connective tissue, the neuroglia, blood-vessels, and lymphatics. Its disorders involve one or more of the above tissues.

The following is a list of the forms of disease which affect the nervous system:

1. Malformations; incomplete development, or agenesis; defective development, or dysgenesis.
2. Hyperæmia, anæmia, hemorrhage, œdema, and arterial and venous diseases.
3. Inflammations.
4. Degeneration and atrophy, softening, sclerosis, gliosis.
5. Tuberculosis and syphilis.
6. Tumors and parasites.
7. Nutritive and functional disorders, including disorders associated with metabolic and glandular defect, such as acromegaly and exophthalmic goitre.

Inflammation.—The pathology of most of the above types of diseases will be given elsewhere, and does not call for discussion here. It is, however, of the utmost importance that the student have a clear understanding of the nature of inflammation and degeneration as they affect the nervous tissue. Inflammation is a morbid process which has to deal primarily with blood-vessels, lymphatics, and connective tissue. Inflammation, teleologically, is the reaction of the organism to an irritant. Wherever there is inflammation, there is irritation. The irritant in inflammation is practically always the product of microbic action or some irritating product of tissue change. Without some microbic or tissue irritant there can be no inflammation. We make this exception only: that certain chemical substances, such as alcohol, arsenic, and lead, may at times excite a form of inflammation, which is, however, probably, primarily a degenerative or destructive process. Inflammation, when the irritant is removed, tends to subside. It is a regressive, not a progressive, process. Bearing these facts in mind, it will be found that inflammations of the nervous tissues never or very rarely occur without the presence of some mi-

crobe or some destructive process whose irritant products excite inflammatory reaction.

Inflammations may be divided into the (1) exudative and the (2) productive forms (Delafield). The exudative inflammations may be simple; without necrosis, with necrosis; purulent; purulent and necrotic.

1. *Simple exudative inflammation* is accompanied with congestion, stasis, emigration of white corpuscles, and perhaps diapedesis of red cells, transudation of blood serum, and formation of fibrin, the total result being an exudate containing white blood cells, now called pus cells, and fibrin in varying proportions. There is, in some cases, no destruction of tissue; and on subsidence of the inflammation the tissue returns to its normal condition. In other cases the inflammatory action destroys some of the nerve tissue.

In *purulent inflammation* there is a great accumulation of pus cells and less relatively of fibrin.

If the tissue is destroyed, it is a *purulent and necrotic inflammation.*

In some exudative inflammations there is increase of connective tissue from the start, and the process continues till the inflammation subsides. Most exudative inflammations are acute or subacute. *Inflammatory œdema* is a form of exudative inflammation.

2. *Productive* or *proliferative inflammation* is a process in which there are little congestion and exudation, while new connective tissue is slowly formed. Productive inflammation is usually chronic; tuberculous and syphilitic processes are varieties of productive inflammation.

The principal poisons which may cause chronic productive inflammations are alcohol, lead, and arsenic. Certain irritating autotoxæmic agents, such as occur in gout, rheumatism, diabetes, and states of inanition, appear able at times to cause productive inflammations.

Classification of Inflammations.

Form.	Cause.	Example.
Simple exudative, with or without necrosis.	Microbic or toxic.	Meningitis. Poliomyelitis.
Purulent, with or without necrosis.	Microbic.....	Meningitis and encephalitis.
	Microbic.....	Acute purulent myelitis.
Productive or proliferative..	Microbic or toxic.	Chronic meningitis.
		Leprous neuritis.

Degenerations and Scleroses.—By degeneration is meant in pathology a gradual death of the nerve cells and fibres, or in other

words of the parenchyma of the organ. The cells swell up, become granular and fatty, and then either break up and become absorbed or enter into a condition of a dead coagulum (coagulation-necrosis). Degenerations may be acute or chronic, primary or secondary.

Acute degeneration causes a condition known as *softening* or *necrosis*. It is due to cutting off of vascular supply, direct injury, and to necrotic and inflammatory poisons. Acute degeneration may be followed by a reparative process, which is called a reparative or reactive inflammation, and which ends perhaps in producing a cicatrix or sclerosis.

Chronic degeneration is accompanied and followed by a proliferative process which results in the production of connective tissue and sclerosis.

Sclerosis is a process of connective-tissue proliferation, as a result of which the normal or injured parenchyma is supplanted by fibrous tissue. The word sclerosis is usually employed in describing degenerative diseases, though it indicates the result rather than the primary nature of the process. In the nervous system there is often an increase or proliferation of neuroglia tissue in the processes of degeneration. Exactly how large a factor this is cannot yet be said.

A *primary degeneration* is one in which the process is due to inherent defect in nutrition or to some poison acting directly on the cell or fibre.

A *secondary degeneration* is one that is due to a cutting off of nerve fibre or cell from its trophic centre, or to an injury or shutting off of its vascular supply. Ordinarily, in speaking of secondary degenerations one refers to those due to the first-mentioned class. Practically, primary and secondary degenerations often occur in the same disease.

Degenerations.	Forms.	Examples.
Acute and Chronic	Primary	Myelomalacia.
		Progressive muscular atrophy.
		Locomotor ataxia.
	Secondary and mixed	Chronic myelitis.
		Secondary lateral sclerosis.

Degenerations are caused by certain poisons, such as arsenic, phosphorus, lead, and the poisons of infectious disease. Degenerations also result from obliterating arteritis, such as occurs in old age or from humoral poisons. Degenerations sometimes are due apparently to an inherent defect in the cell nutrition—a premature death of it; also to causes yet unknown. The question as to

whether certain scleroses are forms of productive inflammation or of chronic degeneration is one that has been much debated in the past. It is quite certain now that the so-called chronic inflammations of the nervous centres are really degenerative processes, and that the primary trouble is in the parenchyma, and not in the connective tissue.

Gliosis.—It is contended by some French pathologists (Chaslin, Dejerine) that some of the chronic degenerative diseases are the result of a proliferation of neuroglia, not of connective tissue. This process is called gliosis. Its existence is not yet satisfactorily established.

Nutritive and Functional Disorders.—Under this head are included defects due to disorders of the blood and blood glands, to defects in metabolism, to poisons, extrinsic and autochthonous, and to local diseases.

In conclusion some fundamental peculiarities of the nervous tissue may be noted here.

Nerve cells once destroyed never develop again.

The same is true, though not so absolutely, for the nerve fibres running in the central nervous system. Peripheral nerves may grow again when cut or destroyed. They always grow *from* their trophic centre. Nerve tissue in brain, cord, or periphery can never be sutured so that it will functionally unite by direct union. There are a few apparent exceptions.

A further peculiarity of nervous tissue is that it is dependent for its integrity upon two things, blood supply and trophic influences. The nerve cell is solely dependent on a proper supply of blood, and dies when this is withdrawn. But the neuraxon is more dependent on the trophic influence of the cell of which it is a prolongation. It dies when cut off from its cell, but it can get along for a time with but little direct blood supply. On the other hand, if the neuraxon is injured it reacts on the cell, leading to a partial but curable degeneration of the cell body.

CHAPTER IV.

GENERAL SYMPTOMS.

When the nervous system is disordered it produces various symptoms, which are classified and receive names according to the parts affected and the kind of change present. The general name given to any kind of morbid nervous state is *neurosis* and the general name for any morbid mental state is *psychosis*. When the neurosis affects the motor sphere, whether in the brain or cord or nerves, it is a motor neurosis, or, more technically, a kinesio-neurosis; when the sensory parts are disordered we have a sensory neurosis, or æsthesio-neurosis. In the same way we have trophic, thermic, vasomotor, and secretory neuroses.

The symptoms of nervous disease are further divided in accordance with the kind of disturbances present. Now a function can only be disordered in three ways. It may be exaggerated, lessened, to the point perhaps of entire loss of function, or it may be perverted. In order to indicate this certain Greek prefixes are used. They are "hyper," which means excess; "hypo," meaning diminution; "a" or "an," indicating entire loss; and "para," meaning perverted. Thus we have, for example, hyperæsthesia, or excessive sensibility; anæsthesia, or loss of sensibility; and paræsthesia, which means perverted sensibility.

Finally, nervous symptoms are often spoken of as objective or subjective. The former are those symptoms which can be seen or directly noted by the physician without depending on the patient's statements. The subjective symptoms are those which are felt by the patient, but give no outward sign. Thus headache is a subjective symptom, paralysis is an objective one.

So far we have been grouping together only like kinds of symptoms; but it happens that many nervous *diseases* may have quite different kinds, some being motor, some trophic or sensory. Thus nervous diseases practically are to a considerable extent classified simply on the basis of the part of the nervous system diseased; and we have spinal-cord and brain diseases, gastric and sexual neuroses, and so on.

Nervous *symptoms*, however, are always grouped together in

accordance with the physiological function disturbed. So that we have the following tabulation (see also Fig. 19):

1. Mental and cerebral, forming psychoses.
2. Motor and reflex, forming kinesio-neuroses.
3. Sensory, forming æsthesio-neuroses.
4. Trophic, forming tropho-neuroses.
5. Vasomotor and thermic, forming angio-neuroses and thermo-neuroses.
6. Secretory, forming secretory neuroses.

Combinations of these groups of symptoms may affect various organs. They are called mixed neuroses. Combinations of mental and nervous symptoms form psychoneuroses.

The particular symptoms which nervous diseases cause will be described and recorded under the several heads given above.

1. The *mental symptoms* include all those found in insanity, idiocy, and imbecility, and will not be given in detail here. The common symptoms met with by the neurologist are mental irritability, depression, emotional excitement, morbid fears, volitional weakness and lack of self-control, persistent or fixed ideas, weakness of memory and of power of concentration, and a tendency to hypnotic and somnambulistic states.

Certain symptoms due to disturbance of brain function are often called *cerebral*, as distinguished from mental. They are: vertigo, disorders of equilibrium, insomnia, somnolence, stupor, coma. Headache, head pressure, and similar feelings are also often described under the head of cerebral symptoms.

2. *Motor Symptoms.*—The symptoms of disordered motility are as follows:

A. Symptoms of exaggerated motility, or hyperkineses.

Tremor: *a*, fibrillary; *b*, tremor proper: intentional, passive, constant.

Convulsions: tonic, clonic, co-ordinate.

Choreic and choreiform movements, athetosis.
Muscular tension and contracture.
Forced and associated movements.
Exaggerated reflexes and clonus.

B. Symptoms of lessened motility.
Paralysis and paresis, amyasthenia.
Loss of reflexes, superficial and deep.

The particular characteristics of these different symptoms will be best shown in the description of the special diseases, but a brief account will be given here.

Tremor is the result of a disorder in the tonic innervation of muscles. Muscles are kept normally in a state of slight tension by rhythmical impulses passing down at the rate of about twelve per second. When the rhythm and force of these normal impulses are interfered with we have tremor. The simplest form of tremor is one in which the normal tonic impulses have an apparently exaggerated force. This causes a fine tremor of eight to twelve vibrations per second. When there is an interruption to some of the impulses we have a coarse tremor. Here the vibrations are five to eight per second. It is caused by a partial or complete dropping out of the alternate impulse. Various technical names are used in describing the tremors. We have the fine and coarse, as described.

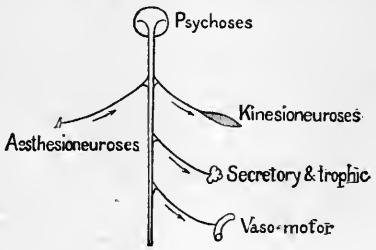


FIG. 19.—DIAGRAM ILLUSTRATING THE PRINCIPLE OF THE CLASSIFICATION OF NERVOUS SYMPTOMS.

Intention tremor is one that occurs on voluntary movement, and is opposite in kind to the passive tremor or tremor of rest, which decreases or ceases on voluntary effort. Tremor is sometimes of a coarse, jerky, and inco-ordinate character, and these words are then used to indicate it.

Fibrillary tremor is a fine twitching of the individual strands or parts of muscles, and occurs usually when they are wasting from lack of neuro-trophic influence.

Convulsions consist of abnormal and exaggerated muscular contractions occurring in rapid succession. Convulsions may be *clonic*, *i.e.*, the muscles rapidly and alternately contract and relax in an exaggerated and irregular way; or they may be *tonic*, *i.e.*, contracted steadily and continuously. When a tonic muscular contraction is painful it is called *cramp*. Convulsions may be co-ordinate. In this case the patient moves the limbs and body in a more or less purposeful way. He throws himself about the bed, jumps, kicks,

strikes, tears the clothes, etc. Convulsions are usually accompanied with loss of consciousness.

Choreic movements are sudden jerking, twitching movements of different groups of muscles. The movements are purposeless and are not under control of the will. *Convulsive tic* is a form of choreic movement confined to certain groups of muscles which work together for a common purpose, like those of the face, or eyes, or larynx. The movements in the "tics" are more definite in character and are limited to muscles physiologically grouped for a definite function. Thus we have tics of the muscles of expression, or of respiration, or speech, or locomotion.

Athetosis is a name given by Hammond to a peculiar form of movement characterized by slow, successive flexion, extension, pronation, and supination of the fingers and hand and arm, or of analogous movement of the toes and feet. The motion rarely ceases in

Fig. 20.—The Hand in Athetosis (Strümpell).

waking hours except for a short time. The contractions are forcible, steady, and even, and sometimes painful. The hand assumes characteristic positions (Fig. 20).

A *contracture* is a tonic muscular spasm of long duration, *i.e.*, days or months. A contracture may be functional or organic; and in order to test this, one must find whether it ceases during sleep or under an anæsthetic; if so, it is functional (see Hysteria).

In forced movements the patient suddenly and involuntarily is thrown forward, sideways, or whirled about in various ways.

Associated movements are those which occur involuntarily in a limb or muscle at rest when the corresponding limb or muscle is moved on the opposite side. Thus in hemiplegia the movement of the normal arm may excite a movement in the one paralyzed. The patient is given a piece of chalk in each hand, and each hand is placed upon a blackboard lying on the table; attempts at drawing lines with the sound arm cause movements of a similar kind, but less perfect, on the paralyzed side.

Paralysis or akinesis is a loss of motor power. Monoplegia is a condition in which one limb is paralyzed; hemiplegia one in which one-half the body is paralyzed; and paraplegia one in which the two lower limbs are affected. Sometimes a double hemiplegia or diplegia occurs. The term paralysis is sometimes used to indicate loss of any kind of function, as paralysis of sensation or secretion.

Paresis is a term used to indicate a partial paralysis. It is not to be confounded with the term general paresis, which is a form of insanity.

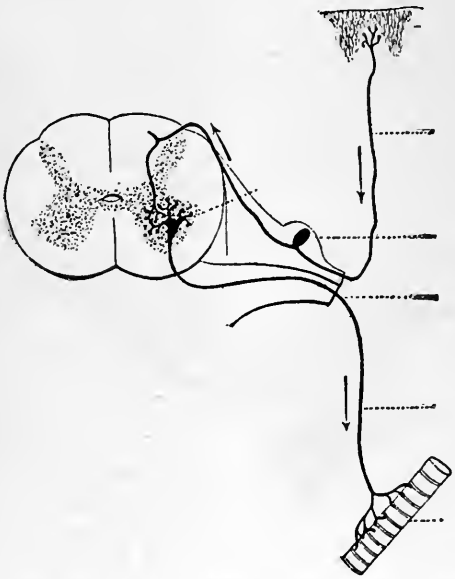


FIG. 21.—SHOWING THE COURSE OF THE IMPULSE IN A SIMPLE REFLEX ACTION.

The Reflexes.—When an impulse started in an afferent nerve reaches the spinal cord or medulla and is thence reflected upon an efferent nerve, the result is called a simple *reflex action* (Fig. 21). The process is an involuntary one. It ordinarily occupies one-tenth to one-twelfth of a second. The afferent nerve may be an ordinary cutaneous sensory nerve, or it may be a special nerve whose function is to excite reflex action. These latter nerves are called excito-reflex. This kind is principally supplied to the viscera.

In neurology we have to do with four kinds of reflexes:

The skin or superficial reflexes.

The tendonous or deep reflexes.

The visceral reflexes.

Idiopathic muscle reflex.

All these may be exaggerated, lessened, or absent. Further description of these reflexes will be given under the head of diagnosis.

3. *Sensory Symptoms.*—The sensory functions include all those belonging to the nerves of general and special sensation. Sensory nerves have a part in reflex action and in the inhibition of motor and other functions. The nerves of special sense when deranged show various phenomena, which will be described in more detail later. In general there may be depression or loss, increase or perversion of their function. In accordance with this we have:

Anæsthesia which is a loss of tactile sensibility.

Analgesia, a loss of sensibility to pain.

Thermo-anæsthesia, a loss of sensibility to temperature. There may be loss of cold-sense or of heat-sense, or, as is usually the case, of both senses.

The term anæsthesia is often used with a general meaning to indicate loss of all forms of sensibility. Anæsthesia in this sense is a symptom referred to the skin, bones, mucous membranes, special senses, or viscera. The muscles have two kinds of sensibility, a sensibility to pain and a special muscle sense. Anæsthesia of the pain sense of muscle is called loss of muscular sensibility or muscular analgesia. Anæsthesia of the special muscle sense is one of the factors in causing a symptom known as ataxia.

Ataxia is a symptom due to loss of the special sensibility of the muscles, articular surfaces, and tendons. This special sense informs the individual of the degree and strength of muscular movements, and by it definite and co-ordinated movements are made possible. The weight of objects and position of the limbs are also determined by it. In *static ataxia* there is loss of the power to preserve perfectly the equilibrium when standing. It is due to the form of anæsthesia just referred to. In *locomotor* or *motor ataxia* there is loss of power to co-ordinate the limbs properly in motion. In these conditions there is also usually a loss of power to appreciate weights or the position of the limbs. The term *muscular anæsthesia*, however, is often used to indicate these latter symptoms. *Cerebellar ataxia* is a form of inco-ordination due to disease of the central organ of equilibration, viz., the cerebellum.

Astereognosis is a symptom indicating a loss of ability to appreciate the form or shape of objects felt.

Hyperæsthesia is an excessive sensibility to touch, contact, and other stimuli.

Hyperalgesia is excessive sensibility to pain, and is nearly identical with tenderness.

Dysæsthesia is an abnormal sensation, such as a "thrill" or feeling of discomfort produced by ordinary tactile or painful impressions.

Paræsthesia is a term applied to all the morbid general sensations except pain. The paræsthesias include such feelings as numbness, prickling, formication, flushing, burning, itching, coldness, tickling, feelings of weariness, exhaustion, various peculiar visceral sensations. Ordinarily in speaking of paræsthesiæ, however, we refer to such feelings as numbness, prickling, and creeping.

Delayed sensation is a symptom in which an appreciable time exists, usually one or more seconds, between the time of applying a stimulus and its appreciation in consciousness. Normally a tactile sensation can be felt and responded to in less than one-tenth of a second.

Transferred or *referred* or reflex sensations are those in which the irritation is made at one point and felt at another. Thus an irritation in the stomach causes a pain felt in the forehead. The whole class of so-called reflex pains are really transferred sensations, since in reality there is no reflex action in the process, as will be seen later. *Allochiria* is a peculiar form of transferred sensation, in which an irritation applied on one side of the body is referred to a corresponding point on the opposite side.

4. *Trophic Disorders.*—These are called tropho-neuroses. They consist, so far as relates to neurology, chiefly of hypertrophy and atrophy of nerves, muscle, cutaneous and mucous tissues, joint degenerations, and various skin eruptions. The tropho-neuroses, if they affect joints, are called arthropathies; if muscles, atrophies, hypertrophies, and dystrophies; or if with atrophy there is a great substitution of fat the condition is known as lipomatosis. When nerves are affected there results degeneration. Tropho-neuroses of the skin produce various symptoms, such as herpes, pemphigus, and other eruptions, pigmentation, leucoderma, alopecia, and bedsores.

5. *Vasomotor and Secretory Symptoms.*—The nerves supplying the blood-vessels and secreting glands work together and are usually disordered together. Separate disturbances of the vessels and glands, however, occur. *Angio-neurosis* is the term given to disorders of the vasomotor centre and nerves. *Angio-spasm* is a condition in which there is increase of vasomotor tone and spasmodic contraction of the muscular coats of the arteries. *Angio-paralysis* represents the opposite condition. Such disorders affecting the skin are shown by pallor and coolness or by flushing and heat. *Angio-ataxia* is a condition of variability and irregularity in the tonus of the blood-vessels.

6. The *secretory neuroses* affect the functions of the skin, mucous membranes, and special glands. *Hyperidrosis* is an excessive sweating. *Anidrosis* is excessive dryness. *Paridrosis* is a perversion

of secretion in which peculiar odors or colors are noted. *Hæmidrosis* is the term applied to bloody sweating.

The secretions of the internal organs are controlled by nervous influences, and their special disturbances often form part of the symptoms of nervous diseases. Thus we have watery diarrhœa in Basedow's disease, and a peculiar membranous discharge from the bowel in asthenic states.

The blood glands, and particularly the thyroid and pituitary gland, have perversions of function which lead to serious nervous symptoms, which will be described under the head of exophthalmic goitre and acromegaly.

CHAPTER V.

DIAGNOSIS AND METHODS OF EXAMINATION.

The diagnosis of a nervous disease may be simply a clinical one; that is to say, one may recognize it as belonging to a certain known and definite group of symptoms. Thus in recognizing the phenomena of epilepsy, one makes a clinical diagnosis. In other cases, and especially in all organic nervous diseases, the physician must make in addition a local, and then a pathological diagnosis. That is, we must determine the seat and nature of the disease.

A diagnosis is made by first getting all the obtainable facts in the patient's past history, then by learning from him all his subjective symptoms, and finally by making an examination according to the technical methods to be here described. In examining a patient, it is imperative that a careful search for diseases outside the nervous system first be undertaken. Then the morbid nervous phenomena should be investigated. The physician should make it an invariable rule to make this examination in a certain fixed and systematic manner. The best method is first to get the family and personal history, and then to go over the mental, cerebral, and special nervous functions serially in the way indicated under the description of general symptoms, thus: Examine—

1. Physiognomy, general condition of nutrition, complexion, physical defects (stigmata of degeneration), gait, station, posture, speech.
2. Mental and cerebral symptoms.
3. Motor and muscular symptoms, including muscular and joint atrophies, electrical reactions, and the reflexes.
4. Sensory symptoms, general and special.
5. Vasomotor, trophic, and secretory.
6. Visceral centres.

In investigating the family history, it is often necessary to make very direct and probing inquiries, for patients are, as a rule, inclined to forget or ignore the existence of nervous and mental disease among relatives. The existence of consumption and inebriety, epilepsy and syphilis, in the direct line are very important facts; so also are those concerning birth. The patient should be questioned closely as to his previous diseases, especially syphilis; also as to his

habits in relation to sexual indulgence, indulgence in alcohol, and smoking. In women, the tea habit should be inquired into. The patient may be allowed to tell his own story first. Proper queries should be put to supplement this, and finally the patient should be asked to state those symptoms which to his mind are main and dominant.

We will now go over the above points in detail.

1. The physiognomy, complexion, and general nutrition are first noted. Many nervous disorders are compatible with a very healthy appearance, and patients often make the introductory apology, "I don't look like a sick person." An anxious look, restless manner, and excited or diffident speech, however, often show something wrong. The nervous trouble is usually serious in reverse proportion to the voluble anxiety of the patient to make his condition exactly understood. The character of the gait may reveal at once the nature of the malady. The dropped foot and flaccid swing of the leg in poliomyelitis and neuritis, the stiff shuffling march of paraplegia from myelitis, the waddling movements of juvenile muscular dystrophy, and the bent head and careful stamp of locomotor ataxia are almost of themselves diagnostic:

Et verus incessu patuit morbus.

The speech also often betrays the malady. The physician soon gets to recognize not only the striking symptoms of aphasia, but also the weak piping of paralysis agitans, the stumbling enunciation of paresis, and the peculiar dysarthrias of multiple sclerosis and bulbar palsy. As a rule, the occurrence of speech difficulties in adults is significant of organic and often serious disease.

I regard it of much importance that in the chronic and constitutional nervous maladies careful note be made of the marks of degeneration. The nature of this condition has already been described under the head of hereditary causes of nervous disease. As already stated, degeneration means a marked and morbid deviation from the normal standard of the race. The existence of degeneration implies an imperfect or an unbalanced development of the body. The condition is usually shown in some nervous or mental defect in the individual, and degeneracy, as ordinarily understood, implies a neuropathic or psychopathic state. But degeneracy may also mean only a lessened vital resistance to certain forms of infection or injury, as, for example, in persons of a tuberculous tendency, who often have marks of degeneracy. However, in ordinary use of the term it applies to those who have inherited nervous and mental weaknesses. The degenerate shows certain marks which are called the stigmata of degeneration. These are of three kinds: anatomical,

physiological, and mental. I have space to give only the more important.

Anatomical stigmata:

Cranial anomalies, *e.g.*

- Asymmetry of cranium.
- Microcephalus.
- Peculiar shape of skull, trigonal, scapho-cephalic, plagiocephalic.
- Facial asymmetry, and excessive prognathism.
- Large jaws.
- Deformities of the palate and uvula, including high narrow arch and the torus palatinus.
- Anomalies of the teeth, tongue, and lips.
- Anomalies of the eyes: narrow palpebral fissure, muscular insufficiency, excessive astigmatism, nystagmus.
- Anomalies of the ears: badly placed, ugly shapes, asymmetry, adherent or lobeless ears, markedly conchoidal ears.
- Anomalies of the limbs, genital organs, and body generally.
- Anomalies of the skin, excessive hairiness, or absence of hair.

Physiological Stigmata.—Tremor, tics, nystagmus, and hereditary defects in the muscular system leading to atrophies. Excessive or defective sensibility of the cutaneous and special senses, defects in speech, perversions of the sexual and other instincts are to be classed here. A diminished resistance to nervous and emotional strain is a most frequent physiological mark of degeneracy.

Mental Stigmata.—These include all those factors that make up the erratic, unbalanced, and morbidly emotional individual. The specially morbid note in these persons, as Peterson says, is an excessive egotism, an intense self-consciousness, often with peculiar disturbances of the sense of personality. Mental degeneracy is often associated with great special aptitudes, even genius, and is quite compatible with sanity and a fair degree of health.

Of the foregoing the most important of the anatomical stigmata are deviations in the symmetry and shape of the skull, defects in the palate and under jaws, badly shaped ears, badly set teeth, and a generally weak and badly developed body. Stress is laid upon the skull because its development corresponds with that of the brain. The palatal stigmata are in general those which make the cavity of the mouth smaller, it being the fact that the mouth cavity increases in size as we ascend the vertebrate series (Peterson). Abnormal palates are found in about ten per cent. of normal people (Charon) and in from forty-six to eighty per cent. of degenerates. The high narrow palate is one oftenest seen by myself. The torus palatinus or longitudinal ridge on the hard palate is significant if it

is well marked. The importance of defective ears is based upon comparative observations. They are found in from twenty to sixty-four per cent. of degenerate persons.

While many of the stigmata have no significance in themselves, yet a combination such as impresses the observer with its preponderance is of great importance, for neuroses or psychoses developed among this class have a much more unfavorable prognosis. It is especially among neurasthenics, epileptics, severe forms of hysteria, and in the insanities that these signs are to be looked for and studied. Among normal men about two or three anatomical stigmata are often found; among lunatics, criminals, abortive types of paranoia, and primary forms of neurasthenia, the number is much greater.

The accompanying table will be of help in making the investigations relating to the cranium (page 43).

To understand it, it is necessary to describe the skull landmarks, and to give briefly the classification and terms used by anthropologists and alienists in describing the dimensions and shape of the skull.

Dimensions and Shape of Skull—*General Classification.*—Anthropologists make a general classification of skulls into:

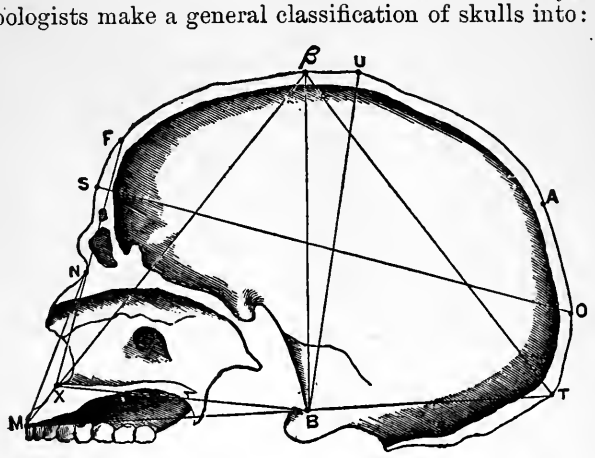


Fig. 19.—*M*, *β*, *T*, The Triangle for Ascertaining the Empirical Greatest Height (Benedict).

The dolichocephalic, in which the antero-posterior diameter is to the transverse as 100 is to 75 or less; the brachycephalic, in which the length is to the breadth as 100 is to 80 or more; the mesocephalic, in which the length is to the breadth as 100 is to 75 to 80. The physiological limits of variation in the ratio of length to breadth are from 100 to 70 to 100 to 90. The dimensions and shape of the skull vary with age, sex, individual, race, and with certain pathological conditions and artificial deformities. In general, however, the variations in the shape and size of the skulls of

Table of Cranial Measurements, in Centimetres.

	Adults.		Physiological Variation.	New-born.		End of 1st yr.		1st to 7th yr.		10th year.		7th to 18th years.	16th to 24th years.	End of 12th yr.	
	M.	F.		M.	F.	M.	F.	M.	F.	M.	F.				
1. Greatest circumference	52.0	50.0	48.5 to 57.4	34.0	34.0	42.0	42.0	34 to 46	....	49	47	46.0 to 49.5	49.5 to 52.25	....	Taken around glabella and maximal occipital point.
2. Binauricular arc	32.0	31.0	28.4 to 35.0	20.0	20.0	25.5	25.0	.....	27	.....	....	.....	.....	30	Measured from *B* over through bregma to *B*, or opposite ext. and. meatus.
3. Volume	1,500	1,300	1,201 to 1,751	385 to 450	....	700 to 1,000	....	.....	....	1,300	....	.....	.....	....	} Benedict and } Huschke.
4. Naso-occipital arc	32.0	31.0	28 to 38	22.0	22.0	28.0	28.0	.....	....	.....	....	.....	.....	....	$N\beta T$.
5. Naso-bregmatic arc	12.5	12.0	10.9 to 14.9	7.7	7.7	.....	10.0	.....	....	.....	12	.....	.....	....	$N\beta$.
6. Bregmat. lamb. arc	12.5	11.9	9.1 to 14.4	9.0	9.0	.....	10.0	.....	....	12	....	.....	.....	....	β to A.
7. Lamb. occipital arc	7.0	7.1	.....	.....	....	.....	...	.....	....	.....	....	.....	.....	....	A to T.
8. Antero-posterior diameter	17.7	17.2	16.5 to 19.0	.....	....	.....	....	.....	....	.....	....	.....	.....	....	S to O.
9. Greatest transverse diameter	14.6	14.0	13 to 16.5												
10. Cephalic or length-breadth index	82.2	83.8	76.1 to 87												
12. Facial length	12.37	.....	10.5 to 14.4	.....	....	.....	....	.....	....	....	....	.....	.....	....	From *N* to lowest point of chin.
13. Empirical greatest height	13.3	12.3	11.5 to 15	.....	....	.....	....	.....	...	.....	....	.....	.....	....	The empirical greatest height, $B\ \beta$, is obtained by measuring the sides of the triangle $M\ \beta\ T$.
Dolichocephaly	.....	.....	70.0 to 74.9												
Mesocephaly	.....	.....	75.0 to 79.9												
Brachycephaly	.....	.....	80.0 to 84.9												
Eurycephaly	.....	.....	over 100 to 150												

These figures are too low for children of the educated classes in this country by at least one centimetre.

healthy adults of European and American races are fairly uniform.

Variations Dependent upon Age.—The proportions of the skull change most considerably in the first year, and continue to change up to the fourth year. After that, modifications are slight in amount and appear more slowly. By the end of the seventh year the skull has nearly reached its full size (see table), more nearly in girls than in boys. The chief measurements during childhood are given in the table. The protuberances and ridges are less marked in children.

The female skull is larger posteriorly, is broader, lower, with higher orbital diameter; often it has no glabella, no super-glabellar depression, and is less well marked as to its ridges, prominences, and sutures.

Variations as Regards Race.—The length-breadth index and other cranial indices and the volume are the only racial differences so far extensively studied. Even these are too indefinite factors to be of any practical value. In general, we may say that the dolichocephalic or long-headed are: the English, Irish, Scandinavians, negroes, 73; Arabs, 74; Chinese, 76. The brachycephalic or broad-headed are: the Germans, 81; Russians, Turks, 81. The mesocephalic or medium-shaped heads are: the American Indians, 79; Hollanders, Parisians, 79.

The Variations Dependent upon Artificial Deformities, Accidents, Perversions of Growth and Development, and upon Disease.—There are certain more or less pathological variations in the shape of the skull, due to a premature ossification of a suture, or arrest of development in a centre of ossification, or to a hyperplasia or aplasia of a part of the skull or of its contents. When one part is shut off from its natural expansion, other parts, as a rule, undergo compensatory development. This principle underlies the pathology of cranial deformities. Those deformities which it would be well to look for are:

The triangular or trigono-cephalic skull; the keel-shaped or scapho-cephalic skull; the acrocephalic or pointed skull; the oxycephalic or steeple-shaped skull; the flat-headed skull; the plagiocephalic or obliquely deformed skull.

Variations in the Neuropathic and Psychopathic Classes.—Variation from the regular type is oftener found in these classes than in the normal, but definite variations corresponding with a special type of disease are not yet made out.

For all ordinary purposes the only instruments needed in examining the cranium are a tape, a strip of lead to use as a conformator, and a pelvimeter.

2. Investigation of Symptoms of Disordered Motility.—In studying the attitude, expression, gait, and speech, some notion of the condition of the motor functions has been obtained. Special

disturbances of the various parts must then be investigated. The patient is made to extend the arms and move them in all possible ways; the face, trunk, and lower limbs are put through all their changes. The degree of paralysis in some groups of muscles can be measured by dynamometers. The ordinary hand dynamometer of Mathieu measures the degree of paralysis in the flexors. It should be graduated accurately in pounds or kilograms.

The average power of pressure on the Mathieu dynamometer is, for an adult, forty to fifty kilograms for the right hand, and three to five kilograms less for the left. A woman has about two-thirds of the power of a man. I have had constructed an apparatus by which the strength of the leg push, *i.e.*, of the extensors of the leg and foot, and the extensors of the thigh is tested. Dr. W. Kraus has devised a simpler instrument. The anterior tibial and calf muscles can also be tested by means of an instrument called the pedodynamometer devised by the late Dr. William R. Birdsall.

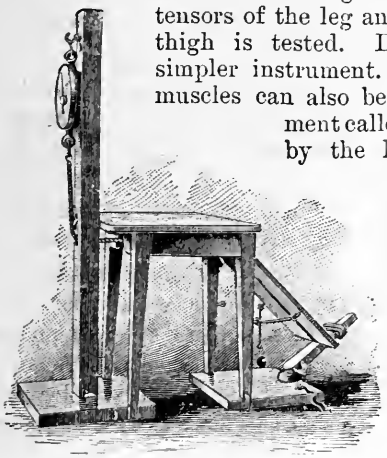


FIG. 23.—LEG DYNAMOMETER.

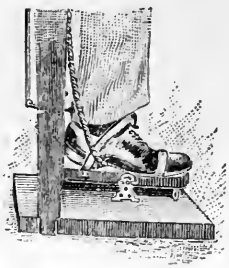


FIG. 24.—FOOT DYNAMOMETER.

A good idea of the degree of paralysis can be got by making the patient take the physician's two hands with his own and squeeze each at the same time. A malingerer or hysteric will often in this way unconsciously press much harder than he is aware. The physician's own ingenuity will suggest various ways of testing the strength of the leg and thigh muscles, such as making the patient rise on one toe, climb upon a chair, push against an object with his foot, etc.

Tremor is tested by making the patient hold out the hands and arms at full length, spreading out the fingers at the same time. To determine whether the tremor increases on volitional movement, give the patient a full glass of water, let him hold it out for a moment, then bring it to his mouth slowly. If the tremor increases with this movement it is called "intention." As a general rule, the tremor of organic disease is increased by volitional movement,

and ceases during rest of the extremity. Functional tremors are usually continuous. In most forms of tremor the hand and arm shake as a whole. In other forms the tremor involves only the fingers or hand or forearm and hand. Such tremor is called segmental. It is especially seen in paralysis agitans. As I have already

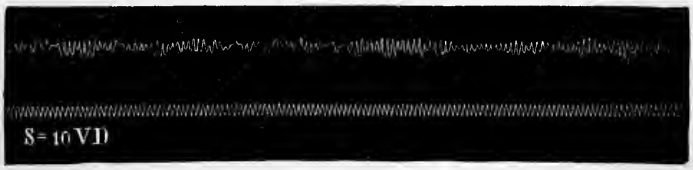


FIG. 25.—DIAGRAM OF A FINE VIBRATORY TREMOR. Ten of the divisions on the lower line equal a second.

said, tremor may be fine or coarse, *i.e.*, four to six or eight to twelve per second. To determine this accurately a special apparatus is needed; but one can with a little experience determine this fairly well by observation alone. Or we can use a sphygmograph, as shown by Dr. F. Peterson. This instrument is fixed firmly on the table, and the tremulous forefinger held lightly against the lever. Coarse tremor is usually a sign of organic disease or of paralysis agitans, but it occurs also in hysteria and grave conditions of alcoholism. Tremor that is hardly observable by the eye can be felt by placing one's hand against the extended fingers of the patient. Tremor of the tongue and lips and facial muscles must be carefully looked for. It is tested by making the patient close the eyes tightly and show the teeth or protrude the tongue. Facial tremor if very marked usually indicates a serious condition of nervous exhaustion, alcoholic poison, or perhaps oftenest of paresis. Tremor of the whole head due to the neck muscles must be distinguished from secondary shaking of the head due to a tremor of the trunk.

Fibrillary tremor, which involves only certain fibres of the muscle, is seen oftenest in the tongue and face and muscles of the extremities. It indicates wasting or exhausted muscles.

Choreic movements, tics, associated and forced movements, and the other forms of motor disturbance can be recognized by simple observation.

Myoidema is a tonic spasm of a part of a muscle near its tendonous attachment. It is produced by a sharp blow upon the muscle near its tendonous insertion. This causes the muscular fibres to bunch up into a small tumor for several seconds. Its presence indicates rapid muscular wasting from exhausting disease.

Idiopathic muscular spasm is a phenomenon of a similar nature. When the belly of a muscle is struck with a dull instrument, a

welt of contracted muscle appears and lasts several seconds. It indicates an exaggerated muscular irritability.

The Examination of the Reflexes.—These, as already stated, are of four kinds: (1) the superficial or skin, (2) the deep or tendonous, (3) the visceral, and (4) the muscle reflexes.

1. A *skin* or *superficial reflex* is produced by scratching, tickling, pinching, or irritating the skin with hot, cold, or chemical irritants. The result is a contraction of the muscles supplying the parts near the irritation. The skin reflexes which can be ordinarily brought out are the plantar, cremasteric, epigastric, abdominal, erector spinal, interscapular, palmar, scapulo-humeral, and certain cranial reflexes.

The plantar reflex is produced by tickling or scratching the soles of the feet. This causes usually, when carefully done, a slight flexion of the toes. In many cases there is, however, no response. In irritable persons and children there is a sudden dorsal flexion of the foot, and often a contraction of the inner hamstring muscles. In pathological conditions involving the pyramidal tracts of the cord or even the motor centres and tracts in the brain there is a dorsal flexion of the toe. This is called the *flexor response* or the *sign of Babinski.*

The cremasteric reflex is brought out by scratching the inner side of the thigh or the skin over Scarpa's triangle. It causes a drawing up of the testicle, not of the scrotum alone, on the same side.

The abdominal reflex consists of a contraction of the abdominal recti muscles, caused by irritating the side of the abdomen.

The epigastric reflex consists of a contraction of the upper fibres of the rectus, caused by irritating the skin of the lower part and side of the thorax.

The erector-spinal reflex consists of a contraction of some of the fibres of the erector spinæ, caused by irritating the skin along its outer edge.

The scapular reflex consists of a contraction of some of the scapular muscles, caused by irritating the skin over them.

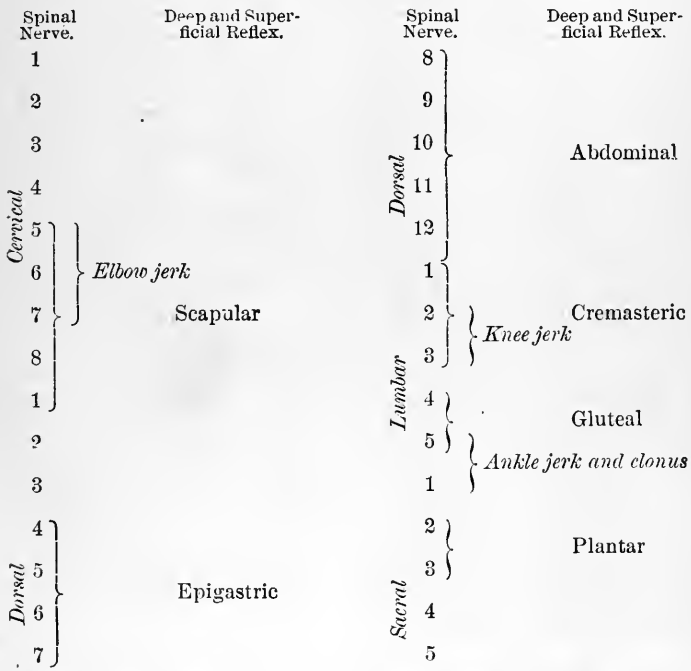
The palmar reflex is produced by irritating the palms of the hands.

The cranial reflexes are the lid reflex, caused by irritations of the conjunctiva or of the retina; the pupillary-skin reflex, which consists of a dilatation of the pupil caused by scraching the skin of the cheek or chin.

The palmar reflex is rarely present in healthy people except during sleep, and in children. The superficial reflexes depend upon the integrity of the reflex spinal arc, and to a less extent upon the

degree of cerebral inhibition. When present, they show that the spinal cord at the level through which the impulses travel is healthy. When absent, they do not necessarily indicate much of anything, for they vary in amount in different persons and at different ages. In cerebral hemiplegia during and for a time after the acute attack, they are generally lessened or absent on the affected side. Later they may be exaggerated.

The levels of the spinal cord through which the impulse travels are indicated in the accompanying table:



Spinal Cord Levels of the Superficial and Deep Reflexes.

The *deep reflexes* are sometimes called *tendon reflexes*, though this is not a strictly correct name, since they can be called out by striking periosteum or muscle as well as tendons. The deep reflex in all these cases is probably a true spinal reflex, though some assert that it is due to the direct effect of the concussion or sudden stretching upon the muscle itself (Gowers), which is in a condition of slight tonus. Those who accept this view speak of the deep reflex as indicating the myotatic irritability or muscular tonus. Either view involves the integrity of a reflex arc.

The deep reflexes are very numerous. The important are:

The patella-tendon reflex or knee jerk. The ankle reflex or

ankle clonus. The wrist reflex. The triceps-tendon reflex or elbow jerk The jaw reflex or chin jerk. The light (or pupillary) and accommodation (or ciliary) reflexes.

The patella reflex or knee jerk consists of a sudden contraction of the quadriceps femoris, vastus internus, and subcrureus caused by striking the patella tendon when the leg hangs loosely at right

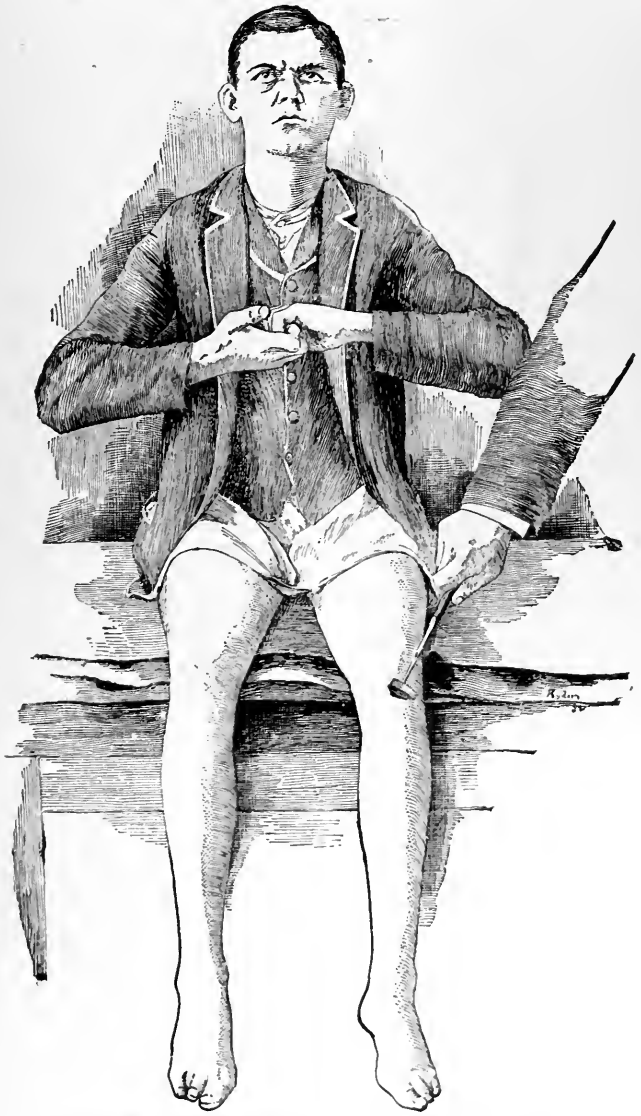


FIG. 26.—GETTING THE KNEE JERK BY RE-ENFORCEMENT.

angles with the thigh. This reflex may also often be produced by striking the lower part of the muscle itself. The activity of this reflex is increased if, at the same time that the blow is struck, a voluntary contraction of some other muscles is made by the patient. Usually the patient is told to pull on his clasped fingers, or tightly shut the hands. This process is called the *re-enforcement* of the knee jerk (see Fig. 26). Such re-enforcement can be caused by irritating the skin and by various sensory or psychic stimuli. The nerve roots involved are those, in man, of the second and third lumbar segments. The peripheral nerve is the anterior crural. The most essential muscles are the vastus internus (Sherrington) and

FIG. 27.—GETTING THE ELBOW JERK.

the quadriceps. The wrist reflex is brought out by striking the wrist tendons while the forearm is supinated and held limply on the hand of the physician. The triceps reflex or elbow jerk is brought out by striking the triceps tendon while the arm is supported and the forearm allowed to hang down loosely at right angles to the arm (Fig. 27). These reflexes occur in normal individuals. The jaw reflex or jaw jerk is brought out by having the patient open the mouth and leave the jaw relaxed. A flat instrument like a paper

cutter is then laid on the teeth of the lower jaw, and if this is struck smartly the elevators of the jaw contract. The light reflex is caused by throwing a bright light into the eye, and the ciliary or accommodation reflex by making the patient look at a distant and then at a near object. The pupil normally dilates in the former case and contracts in the latter. When the light reflex is lost while the accommodation reflex remains, the condition is called the *Argyll-Robertson pupil.*

Ankle clonus is caused by having the seated patient extend the limb and hold it rather firmly in a semiflexed condition. The physician takes the foot by the toe and heel and quickly flexes the foot on the leg. He thus suddenly stretches the calf muscles, and they undergo rhythmical contraction. This phenomenon does not occur in healthy people. It is found in transverse and compression myelitis and in degeneration of the lateral columns of the cord, and it usually indicates organic disease of the cord. A pseudo-clonus sometimes occurs in which there

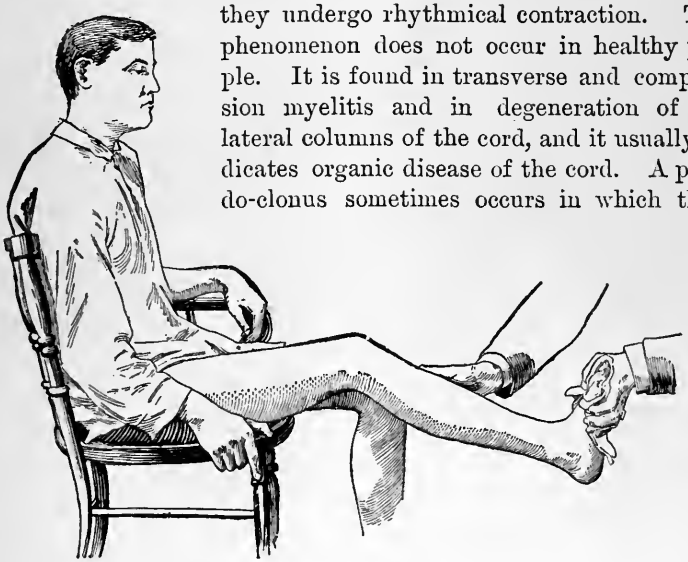


Fig. 28.—Getting Ankle Clonus.

are a few rhythmical contractions on sudden dorsal flexion of the foot, but the contractions soon subside. This is seen in neurasthenia and hysteria.

The deep reflexes, and in particular the knee jerk, for that is the one most easily and often tested, are practically always present in health. They may be decreased, delayed, absent, or exaggerated. Their exaggeration is common and not of special clinical significance. The absence of the knee jerk is of great significance, indicating in persons who have no paralysis of the crural muscles, locomotor ataxia, neuritis, or some toxæmia, such as follows diphtheria or exists in diabetes.

The "paradoxical contraction" is a name given to the tonic con-

traction of the anterior tibial muscles caused by the physician's suddenly flexing the foot on the leg, thus shortening these muscles. This is a rare phenomenon, never found in health, and usually associated with excessive spasticity of the legs.

The Electrical Conditions in Disturbances of Motility.—These cannot be understood without some description of the methods of using electricity, and hence the technique of electrical examinations for purposes of diagnosis will be described under the head of treatment.

Examination of the Disorders of Sensation.—The object of examining the sensory functions is to see if they are exaggerated, perverted, or lost, and to locate the extent of the disturbance. Patients differ greatly in their intelligence and power of description, so that great care must be taken in drawing conclusions as to sensory disturbances. In examining the skin and muscle senses, the patient's eyes should be closed and he should be carefully told to answer promptly whenever he feels the stimulus. It is best to in-

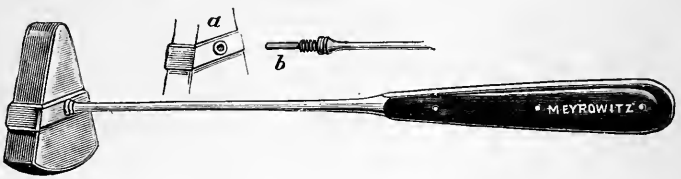


Fig. 20.—Instrument for Testing Tendon Reflexes.

sist that he always reply in the same way, *e.g.*, using the word "now" the moment the sensation is felt. Many ingenious instruments have been devised, and I have described some of them, but for ordinary purposes a camel's-hair pencil and a pin answer very well.

Sensations are of two kinds, general or common and special. A common sensation is one which is referred to the body, and it is subjective in character. A special sensation is one which is referred to the external world, and in particular to the object which causes the stimulus. The pain from a knife cut is referred to the body, and is a common sensation. The coldness felt when a knife blade is laid on the skin is referred to the knife, and is a special sensation. Special senses give us very often objective symptoms, *i.e.*, such as can be noted directly by the physician.

The sensory functions to be examined are:

The cutaneous.

The muscular, articular, and tendonous.

The visual, auditory, olfactory, gustatory, and space senses.

Visceral and general bodily sensations.

The *cutaneous sensations* are: (1) The tactile sense, which includes pressure and contact; (2) the temperature sense, which includes the heat sense and cold sense; (3) the pain sense. The first two are special senses, the last is a general sense.*

To test the tactile sense, blindfold the patient and use the æsthesiometer. This is an instrument with two rather blunt points, which can be separated or approximated. A hairpin or two ordinary pins can be used in its stead. Its use depends upon the fact that the power to appreciate the contact of two points on the skin gradually approximated varies with the tactile sensibility of the patient. The tongue, finger tips, and lips are the most sensitive points. The back, arms, and thighs the least sensitive.

The following table shows the average distance at which two points are appreciated as such by an intelligent adult:

Tip of tongue	1 mm. ($\frac{1}{25}$ in.).	Tip of toes, cheeks, eyelids	12 mm.
Tip of fingers	2 "	Temple	13 "
Lips	3 "	Back of hands	30 "
Dorsal surface of fingers	6 "	Neck	35 "
Tip of nose	8 "	Forearm, leg, back of foot	40 "
Forearm	9 "	Back	60–80 "
		Arm and thigh	80 "

The figures vary somewhat with the thickness or softness of the skin and with the dulness or keenness of the nervous organization. If the distances are double those given above, it may be considered in most cases abnormal.†

The sense of contact, which is a form of tactile sense, is tested by drawing a pencil or a bit of cotton lightly over the skin. The sense of locality or power to localize a point on the skin that has been touched varies with the tactile sense and with the muscular sense. It is tested by placing the finger lightly on a given spot and

* Psychologists deny the independence of the pain sense, and assert that it is only a quality or modification of other senses.

† The tactile sense may also be tested by the writing-method (Rumpf). Figures or letters are written upon the skin with a hard-pointed instrument, and the patient is asked to tell them. The figures drawn are made larger or smaller in accordance with the decrease or increase of sensibility. The following table shows the different sizes as appreciated on the normal skin:

Finger tips	0.5 cm.	($\frac{1}{5}$ in.)	high
Palm	1 "	($\frac{2}{5}$ in.)	"
Neck	1 "	"	"
Cheeks	1 "	"	"
Forehead	1 "	"	"
Arm, forearm, and back of hand	1.5 to 2.5 "	($\frac{3}{5}$ to $\frac{4}{5}$ in.)	"
Scapula	1.5 to 2.5 "	"	"
Calf and sole	3 "	($1\frac{1}{5}$ in.)	"

telling the patient with closed eyes to place his finger on the part touched. He should come within five centimetres of it. In slight degrees of anæsthesia dependent upon disease of the sensori-motor areas of the cortex of the brain this is an important test. Further tests may be made by moving points along the skin and asking the patient to indicate the direction of the motion; or by laying variously shaped objects on the skin and asking the patient to tell their shape or position.

FIG. 30. — COMBINATION ÆSTHESIOMETER.

To test the *pressure sense*, one may use the baræsthesiometer, an instrument made with a spring scale measuring the amount of pressure made. A simpler way is to have the patient rest the hands on a table and then try and determine the weight of different objects. The lightest weight that can be appreciated on the hands or face is one of about 0.02 gram (gr. $\frac{1}{3}$). Differences of light weights of 1 and 5 grams and of 25 and 30 grams are about all that can be ordinarily appreciated by the skin. Much smaller differences, of 0.5 to 2 grams, can be detected if great care is used. Weighted rubber balls may be used in the foregoing test. I prefer to use differently weighted metal bodies, held by a wire. Pressure sense is acute on the forearm and abdomen, where locality sense is feeble; also on the brow, temples, and back of the hand.*

Most of the above tests are not ordinarily needed. With two pins, using the heads, the presence and degree of anæsthesia can be detected and approximately measured.

The *temperature sense* is tested by test tubes filled with hot and cold water, or by using hot and cold spoons, or roughly by breathing and then blowing on the part. A thermo-æsthesiometer may be used. This has a round, flat surface one centimetre in diameter, and contains in its terminals thermometers by which the degree and differences in temperature may be noted (Fig. 30). A small heated or chilled surface is appreciated much less easily than a large one.

* The stereognostic sense is one dependent on the use of the tactile and muscular senses, and is tested by placing variously shaped objects in the patient's hands and asking him to name them.

The indifferent range where objects are felt to be neither warm nor cold is from 27° to 30° C. (80.6° to 86° F.). Fine differences (0.2° to 1.5° C.) are appreciated above the indifferent range. Lower down in the scale, differences from 1° to 1.3° C. (2° to 3° F.) are appreciable. It may be considered a morbid symptom if temperatures of 60° to 65° F. are not felt as cold, or temperatures of 86° to 95° F. are not felt as warm; also if between the ranges of 1° C. (32° F.) and 40° C. (104° F.) differences of 2° C. are not appreciated. A painful degree of sensitiveness to heat or cold sometimes exists. These conditions are called hyperthermalgesia and hypercyalgesia (Skinner, Starr). When the heat or cold is intense, a sensation of pain is felt. Cold pain is produced more easily in some places, such as the elbow, than others, as, for example, the finger tips. Cold pain is produced by temperatures of from +2.8° C. to −11.4° C. Heat pain is produced by temperatures of from 36.3° C. to 52.6° C.

The *pain sense* is tested by pricking the skin with needles or the sharp points of an æsthesiometer. The faradic battery with metal points or a wire brush may also be used. Instruments for pinching the skin and measuring the sensibility by the strength of the pinch have been devised. The power of localizing pain is lessened in proportion to the analgesia.

The *muscular sensibility, i.e.*, the general or pain sensation of muscles, is tested by passing the faradic current through the part.

Pain and temperature sense are usually affected together.

Delayed Sensation.—The time taken for a sensation to be felt and produce a voluntary response is, for—

A touch on the hand, about	0.12	second
" " foot, "	0.17	"
Hearing	0.13	"
Sight	0.16	"
Taste	0.15	"

The tactile sense, as well as the other special and the general sensations, may show a delay in conduction. The tactile sense especially should be tested on this point. The delay may amount to several seconds.

Double Sensations or Polyæsthesia.—When the touch of one point is felt as two or more, the symptom is known as polyæsthesia. *Referred* sensations and *allochiria* are described under Symptomatology. The distribution of the anæsthesia must be determined. The normal nerve supply of the skin is shown in Figs. 31 and 32.

Tests for the Condition of the Special Sense of Muscles, Joints, and Tendons, i.e., for Ataxia.—Anæsthesia of the special sensory nerves of the muscles, joints, and tendons causes ataxia and inco-

ordination. Muscle anæsthesia causes chiefly a loss of *weight sense* or loss of power to determine weights. It is tested by the use of weights suspended by a string so as to exclude pressure sense; also

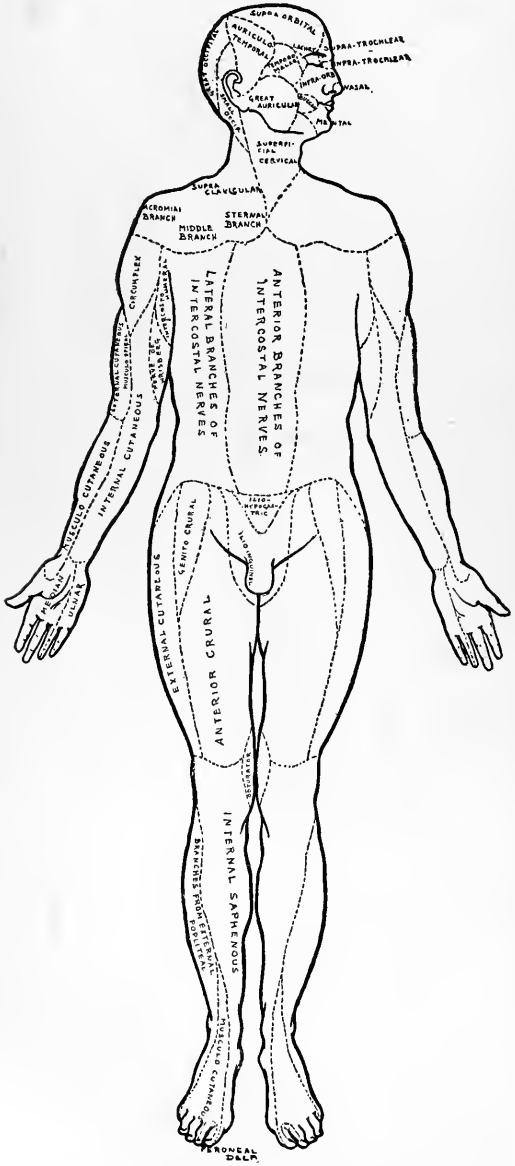


FIG. 31.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE SKIN.

by causing the patient to squeeze a dynamometer up to a certain fixed number.

In articular and tendonous anæsthesia there is loss of posture sense. It is tested by the physician's moving the patient's limbs

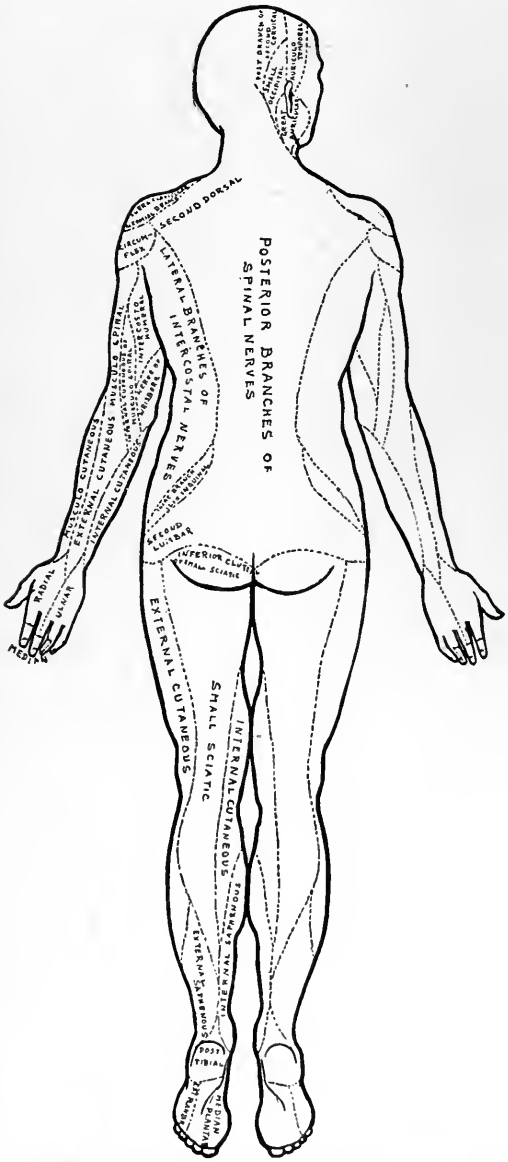


FIG. 32.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE SKIN.

and having the blindfolded patient tell in what direction the movement is made. Or he is told to follow with one limb the movements which the examiner makes with the other.

Muscular, articular, and tendonous anæsthesia usually exist together; there is then ordinary ataxia. Such ataxia shows itself in standing and in locomotion and other voluntary movements. Thus we have a *static* ataxia and *locomotor* or motor ataxia. Static ataxia, or inability to stand (or sit) without swaying or irregular movements, is tested by making the patient stand with the eyes closed and the heels and toes close together. Normally, the head moves not over an inch in this position, and the patient holds the head and body more rigid with the eyes closed than with them opened. In ataxic states the reverse is true, and decided swaying or even complete loss of equilibrium occurs with the eyes closed, or even with the eyes open, and the base narrowed by putting the feet together. This phenomenon is called the "Brauch-Romberg symptom." In static ataxia, muscular and articular sensations are both involved. The degree of this can be accurately measured by the ataxiagraph (Figs. 33 and 34).

FIG. 33.—ATAXIAGRAPH.

With the eyes open a healthy person standing erect with feet together tends to sway forward. The antero-posterior excursion of the head averages 3.7 cm. ($1\frac{1}{2}$ in.), the maximum being 9 cm. ($3\frac{1}{2}$ in.). The lateral excursion averages 1.9 cm. ($\frac{3}{4}$ in.), maximum being 5.4 cm. With the eyes closed the antero-posterior excursion averages 3.4 cm. ($1\frac{1}{4}$ in.), maximum $3\frac{1}{3}$ in.; the lateral excursion 1.9 cm. ($1\frac{3}{4}$ in.), maximum 6.8 cm. ($2\frac{5}{8}$ in.) In other words, the person normally stands a little steadier with the eyes closed, the average excursion being $1\frac{1}{2} \times \frac{3}{4}$ in. with eyes open, $1\frac{1}{4} \times \frac{3}{4}$ in. with eyes closed (Bullard and Brackett).*

No. 1. No. 2.

FIG. 34.—ATAXIAGRAMS. No. 1, Made with eyes open; No. 2, made with eyes closed.

* This is not always the case.

Ataxia of motion is tested by the gait. The patient cannot walk a straight line and cannot walk without watching the floor with the eyes. The arms cannot be moved in a co-ordinate way. With the eyes closed, the patient cannot place the finger on the tip of the nose, or lobe of the ear, or any indicated spot. Ataxia of motion involves especially the articular and tendonous sensations, but not these exclusively. It may be measured by noting how close in walking the patient keeps upon a given line ten feet long; how near he can place the finger upon the centre of a board marked like a target. The patient is placed ten feet away, and made to walk directly at it and place the finger in the centre.

To sum up the foregoing, we have:

	Due to	Tested by
Ataxia.....	Muscular anæsthesia	Weights, etc.
"	Articular and tendonous anæsthesia...	Position of limbs.
"	Combined forms, *e.g.*, static ataxia...	Co-ordinate movements.
"	Locomotor ataxia	Station and gait.

Vision.—The special modes of examination are given under the head of Diseases of the Optic Nerve and Ocular Muscles. The special points which the neurologist must investigate are visual acuity, astigmatism, errors of refraction, limitation of the visual field, exophthalmia, retraction of the bulb, color blindness, the state of the pupil and its reflexes.

Hearing.—The special methods of examination are given elsewhere. The points chiefly to be investigated are acuity, range, bone conduction, aerial conduction, electrical reactions.

Sense of Smell and Sense of Taste.—See cranial nerves.

CHAPTER VI.

HYGIENE, PROPHYLAXIS, TREATMENT.

In the treatment of nervous disease, the physician attempts to relieve distressing symptoms, to secure radical cure, and to prevent return. This calls for various measures, which may be classed under the heads of general hygiene, diet, exercise, climate, hydrotherapy, massage in various forms, electricity, drugs, external applications, and surgical intervention.

General Hygiene.—To secure and keep steady nerves, and to prevent the supervention of organic nervous disease, would require a considerable reconstruction of the present social system. I can only give some hints as to the kind of advice physicians should give to help along the desired end, this being meant more especially for the neuropath. Thus two people of very nervous temperament should not marry. Blood relations of the same temperament should not marry, and families with a psychopathic taint should not intermarry. Children should be brought up to eat slowly a mixed diet, to sleep early and long, to play in the open air, to learn self-control and obedience. Their parents should keep from them all infective fevers. Systematic study and work are good for all children. It is the strain due to defective vision, poor light and ventilation, and unsuitable tasks that hurts the neurotic. Education and occupation are the best kind of builders up of healthy nerves. There are children, however, who cannot follow the ordinary educational lines and who must be specially trained in consequence. The queer and eccentric children with some twist, or precocious talent, need especial care. They usually must be brought up to follow lives on a low mental plane. Too many good farmers and artisans are spoiled by being made poor professional men, or being set up in responsible business positions. Adults need to keep in mind only—moderation, exercise, and the avoidance of a luetic infection. With these they need not fear the use of alcohol, tobacco, tea, coffee, or even occasional irregularities in sleeping and eating. Physical and mental shocks, infective fevers, and poisons are prolific promoters of nervous disease. Syphilis stands out as the most important single factor in producing organic nervous diseases. If it could be removed we would have little if any locomotor ataxia, paresis, or myelitis, and far fewer cases of apoplexy.

Alcohol is a less important factor, but does much to produce mental disease, vascular disease, and hereditary degeneration.

Diet.—For the neuropathic in general, the best diet is a nitrogenous one, but it should contain some fat. Water should be drunk plentifully except by the obese, while the total amount of food should be less than when severe muscular exercise is taken. The best foods are meats, especially fowl; fish, eggs, milk, buttermilk, cocoa, green vegetables, and stale bread with plenty of butter. If there is a tendency to constipation, farinaceous foods and green vegetables may be made the prominent articles of diet in one of the daily meals, and stewed fruit and some alkaline water added. Milk is not a very good food for adults or the aged, except in moderate amount. The drinks of brain workers should be mainly plain and alkaline waters. Alcohol can be taken in moderation by some brain workers without harmful results. It may even secure an increased capacity for work, but this is rarely the case in the American climate.

In persons of an especially irritable nervous system those who are classed popularly as "nervous," neurasthenic, or hysterical, the above rules apply as to a nitrogenous diet, plus as much fat as can be digested. There is a class of nervous persons who of themselves find that they cannot take anything sweet without producing headaches, rheumatic pains, and dyspeptic symptoms. These persons should live on meats, fish with butter, oysters, cream and milk, cod-liver oil, and fat pork. Beef tea with the white of an egg or some peptonoids forms a very nutritious dish. It has been the canon of medicine for many years that animal food must be the soul of the neurotic's diet. Most nervous persons find in addition that green vegetables like spinach agree very well with them. Stale bread can be taken twice a day freely, plenty of butter being used upon it. The dietetic breads from which the starch has been removed are sometimes useful, but are, as a rule, unpalatable, and soon cause disgust.

When a rigid diet is to be laid down, there is no better list for nervous invalids than the following: fowl; beef; mutton and lamb; fish, boiled or broiled; oysters; milk; butter; eggs, raw or soft-boiled; cocoa; graham bread and gluten bread; spinach; Brussels sprouts; string beans; stewed fruits.

Some neurotic persons seem to need a great deal of food, but as a rule harm comes from full diets, and one cannot get strong by stuffing.

The frankly nervous and especially the hysterical patients should not use alcohol at all. Tea and coffee can be taken in very small amount, and best without sugar. The various alkaline mineral waters may be used temperately with impunity, but none of

them have much specific effect in relieving nervousness or curing the nervous temperament.

Water should be drunk between or before meals and a moderate amount at meals. At least three pints of liquid should be taken daily. American neurotics do not drink water enough. They have half-desiccated nerves, and desiccation increases nervous irritability. An exclusive milk diet is indicated in some forms of hysteria, hypochondriasis, and neurasthenia accompanied with dyspepsia. Karell's method is to give four to eight ounces of warm skim milk at 8 A.M., 12 M., 4 P.M., and 8 P.M. The amount is gradually increased. Such diets are, however, only to be kept up for a limited time.

Exercise.—As a prophylactic against nervous disorders, the value of exercise, if taken out of doors, can hardly be overestimated. Brain workers are better for moderate exercise, but they do not need much; and after twenty-five, severe intellectual work can rarely be done by persons in athletic training. Before the age of twenty-five, when the system is exuberant with vitality, hard study and hard physical exercise can be pursued successfully together by some. Persons of a neuropathic constitution are most benefited by regular exercise when it interests the mind. In-door gymnasium exercise with the ordinary apparatus does little good except through the bath that follows it. In many forms of chronic organic nervous disease, exercise is to be prohibited. These will be discussed later.

The best forms of exercise are those which take one out of doors, interest the mind, and call into play the muscles of the chest and arm. Walking fulfils but two and often only one of these conditions. Calisthenics are useful when they interest and are vigorously done. Horseback riding, golfing, and bicycling fulfil best the conditions required for a good form of exercise. And bicycling is the cheaper, more practicable, and generally better liked of these. Lawn tennis, badminton, golf, are all exercises which can be taken up by both sexes and at nearly all ages. Exercise is not so necessary and should be taken in moderation after forty or forty-five.

HYDROTHERAPY.—Hydrotherapy is the science of applying water in the treatment of disease. The modes by which it is used in neurological therapeutics are:

I. General hydrotherapy:
   1. Tonic hydrotherapy.
   2. Sedative hydrotherapy.
   3. Indifferent baths for mechanical purposes.

II. Local hydrotherapy.

1. *Tonic Hydrotherapy.*—For purposes of stimulating nutrition and increasing vasomotor tone we employ cold plunges, the rain

bath or shower, the jet, cold sponging, cold sitz baths, cold sheets, local applications of ice or cold compresses, or cold rubbing, ice bags, brine baths, brief cold packs, and sea bathing.

The cold plunge. The patient fills the bathtub with water at from 60° to 70° F. He then gets in, and at once jumps out and rubs himself vigorously until reaction occurs.

The rain bath and Charcot douche. The patient stands in a tub with the feet preferably in warm water, and allows the cold water to fall on the back and rest of the body for one or two minutes. Or a solid jet of cool water is thrown with force upon the back of the patient, by an attendant who stands ten or twelve feet away (Charcot douche). The cold jet may be alternated with a warm one (Scottish douche).

The cold sheet or drip sheet is used by wringing a cotton sheet out in cold water and wrapping it sudddenly about the patient, who sits or stands with the feet in a tub of warm water. The patient is then dried, put to bed, and vigorously rubbed.

Ice bags are worn upon the spine for one or two hours once or twice, or oftener, daily; or they may be applied for one or two hours at night.

Most of the above measures have a general stimulating and tonic effect.

Cold baths in their various forms, but especially the jet baths like the Charcot douche, are best preceded by a short stay in a hot box until the patient begins to perspire.

The half-bath and wash-off consists of a tub partly filled with water at a temperature of 65° to 70° F. The water only half covers the reclining body. While lying in it, the patient is vigorously rubbed. A cold cloth may be laid on the head. After five to twenty minutes, affusions of colder water are poured over the shoulders. The bath may be made as warm as 80° at first.

Brine baths contain about two per cent. of salt, this being about the amount in sea water—twenty-five pounds to thirty gallons of water. They are given at a temperature of 100° F. for twenty to thirty minutes daily, or cool baths at a temperature of 70° F. may be given for five to ten minutes, the patients exercising meanwhile.

Physiology.—Cold applications produce a local contraction of blood-vessels, followed by dilatation. There are usually increased tissue metamorphosis, increased secretion of urine, increased absorption of oxygen, and increased excretion of carbonic acid. In non-febrile persons, cold applications abstract some heat, but they also stimulate the heat-producing centres, so that the total effect is to increase the heat of the body. Only very cold or prolonged baths lessen heat production as well as excretion of CO_2. Cold baths at first

accelerate and then tend to retard pulse and respiration. Cutaneous sensibility is at first increased. After a cold bath there is a sense of exhilaration and increased muscular power, provided the bath be not too cold or too long continued. The duration necessary to produce a reaction varies with different people, and some weak and sensitive patients never can be made to react. Cold baths systematically taken furnish a kind of vasomotor gymnastics. The neuro-mechanism controlling the blood-vessels becomes more supple, and the tendency to local congestion of the viscera and mucous membranes is prevented. The shower and jet furnish the most valuable means of securing tonic effects in nervous disorders. These are not used with cold water alone. It is often better to apply at first a warm stream at 95° or 105° F., and then gradually lower it, or to apply the hot and cold jets alternately. In this way tonic effects can be obtained even with very feeble persons.

2. *Sedative Hydrotherapy.*—The sedative baths are the lukewarm bath, the wet pack, Turkish and Russian baths, the hot sitz baths, pedal baths, compresses and fomentations, and hot-water bags.

The lukewarm bath is given at a temperature of 95° to 98° F. for ten minutes to half an hour daily. If a slight tonic effect is desired also, the patient should receive an affusion afterward, *i.e.*, basins of cool water at 60° to 70° F. should be poured over the shoulders. The addition of salt or of pine-needle extract is often useful.

The wet pack. A large, thick blanket is spread upon the bed, and upon this is laid a linen or cotton sheet wrung out in cold water, 40° to 60° F. The nude patient lies on this, and the sheet is then smoothly wrapped about him, the head and feet not being included. The sheet is carried between the legs and made to lie evenly in contact with the body. Then the blankets are folded over him, and other blankets may be piled upon this. Sometimes it is well to place hot-water bottles at the feet and a cool compress on the head. The patient lies in this pack for thirty to forty-five minutes and is then rubbed off. A cool affusion may be given first. With delicate patients it is well at first simply to wrap the patient in warm flannels until free perspiration results, then give a cold affusion or wash-off and rubbing.

In Turkish baths the patient is exposed to a temperature at first of 130° or 140° F. for fifteen to thirty minutes, and then to one of 160° or 200° F. for a shorter time. This is followed by massage and a cold affusion or plunge or shower. The effects of these baths are somewhat tonic if not too prolonged. The patient should never go into the hotter rooms until he perspires, and he should select

bathrooms that are well ventilated. *Russian baths* have similar effects, but the bodily temperature is raised to a higher degree in them than in Turkish, owing to the lessened amount of perspiration due to the presence of steam.

Hot sitz baths. The patient sits in water at a temperature of 100° to 125° F. for twenty or thirty minutes. Sometimes mustard is added. Cold sitz baths are given in the same way, and are often useful in sexual neurasthenia.

Hot compresses consist of layers of flannels wrung out in hot water and covered with dry flannels and rubber cloth. They are used to relieve local pains and inflammations. They may be applied over the abdomen for insomnia. *Hot sprays and douches* are used for similar purposes as fomentations. The hot spinal bag is applied at a temperature not above 120° F.

Physiology.—Warm baths, if applied in the form of the moist pack, followed by sponging with tepid water, lessen temperature by increasing heat radiation and conduction. If applied so as to prevent radiation, the bodily heat is raised. Warm baths increase the circulation of the skin, lessen cutaneous sensibility, withdraw blood from the central organs, increase the exhalation of CO_2, but lessen the respiratory activity on the whole. Nitrogenous metabolism is increased by from two to three per cent., and more urea is excreted. Pulse and respiration are increased. Nervous excitement is lessened, and the general effect is to cause sedation and a feeling of languor. The wet pack is a most useful sedative in neurasthenia and insomnia, and may take the place of medicinal sedatives, like the bromides. It should be given three or four times weekly, or for a short time daily. The lukewarm bath ranks next in its sedative efficacy. It is believed that applications of water to the feet and abdomen especially affect the intracranial circulation; given to the thigh and wrists, the pulmonary circulation; cold causing congestion, and heat anæmia, of the distant parts. Cold to the spine is believed to cause at first constriction, and later dilatation, of the thoracic, abdominal, and pelvic vessels; heat has the opposite effect. Hence cold applications are used to relieve cold feet and anæmic conditions of the viscera.

The fact must be borne in mind that cold baths and frequent bathing of any kind debilitate some few persons. Special details of hydrotherapy are given in the appendix.

Massage.—The term massage may be made to include all the manipulations of the body for the purpose of curing disease. The different methods of applying it as classified by Jacoby are:

Effleurage or gentle stroking. The maximum force to be applied here should not exceed the weight of the hand. *Massage à friction*

or rubbing. Energetic strokes with one hand and strong circular or to-and-fro friction with the other. *Pétrissage* or kneading. *Tapotement* or percussion with the fingers, hands, or instruments. *Functional movements*, passive, active, and combined with movements made by the operator. The physician may be reminded that a male operator is a *masseur*, a female a *masseuse*, and that the patient is *masséd.*

Massage accelerates the lymph and venous currents, and thus promotes absorption. It increases at least temporarily the number of red blood cells (Mitchell). It increases the rapidity and force of the heart beat (except abdominal massage, which slows the heart) and helps to relieve local congestions and inflammatory deposits. It presses and stretches the terminal nerve filaments, increases the irritability of motor nerves and the contractility of muscles. It may either increase or lessen the irritability of sensory nerves according as it is applied. Of the various forms of massage, *tapotement* is frequently useful, and is the kind often used in neuralgias. It is applied not only with the fingers and hand, but also by the aid of rubber tubes known as muscle beaters, rubber balls with rattan or whalebone handles, percussion hammers, and various percuteurs.

Regular muscular movements according to a certain fixed schedule are used in the treatment of locomotor ataxia and paralysis agitans. The details of these exercises, known as the method of Fraenkel, will be given later.

Massage is of considerable value in certain forms of atonic neurasthenia and hysteria associated with anæmia, dyspepsia, and feeble circulation, in hemiplegia, in the paralyses of peripheral origin, in functional spasm, especially in some forms of writer's cramp and allied neuroses, in cerebral hyperæmia, insomnia, constipation, and in headache and some neuralgias, especially those about the head, neck, and arm. It is contraindicated in heart disease, arteritis, or when there is danger of dislodging a thrombus.

Climate in Nervous Diseases.—The factors which make up a special kind of climate are: Purity of air; temperature; humidity; sunlight; rarefaction of the air; ozone; wind; electricity; soil; trees; social conditions.

Regarding these points, some facts are very well settled. The air in the country is purer than in cities. The air on the sea and at high levels is purer than in other localities. The temperature above the sea level diminishes about 1° F. for every 300 to 350 feet, and is less the dryer the air. Alterations in temperature are less near the sea and less in the southern hemisphere. The higher the elevation and the colder the air, the less moisture does it contain.

About the factors of ozone and electricity in the air little definite is known.

Climates are classified by Weber into marine low-level inland, and high-level inland. These all have great variations in quality, depending upon their temperature, moisture, etc. As a general rule, warm marine climates and sea voyages are best for neurasthenic invalids of the irritable type. On the other hand, in atonic and anæmic conditions high inland climates are better, at least for a time. Such climates should not be too dry or windy.

In organic degenerative diseases of the nervous system, marine climates and low levels are better.

Germany, the Riviera, the Bermudas, the West Indies, southern Colorado, Arizona, and southern California are favorite places for sending neurasthenic Americans. Camp life in the Adirondacks or other forests is also found most useful.

Electricity in Nervous Diseases.

Physical.—Electricity is assumed to be a material like a fluid, perhaps a condition of the ether itself. It is not a force any more than water is a force, but it produces force by its movements. Electrical phenomena are the result of the strain or stress put upon the electrical fluid (Lodge). Physicists assume that the electrical fluid exists in two conditions, positive and negative, and we speak of positive electricity and negative electricity accordingly. Under ordinary conditions these fluids are united and in equilibrium; but by certain agencies, such as friction, heat, chemism, etc., they can be separated. We assume that the condition of electrical equilibrium is that of zero, and that the earth's electricity is at zero. Positive electricity is raised above, negative electricity pulled below, this zero point. Electrical phenomena result from attempts of the fluids to become equalized or stable again at the zero point, just as the phenomena of heat result from differences of temperature and those of gravity from difference of pressure. The distance to which the electrical fluids are separated from the zero point is spoken of as the difference in "potential." This *potential* corresponds to the term "degrees" in measuring heat. Now, the greater the difference in potential, the greater the effort of the fluids to return to zero. High and low potentials correspond to high or low intensity of heat. *Tension* is the result of the widely separated fluids striving to return to the zero point. It is the same thing as *difference of potential*, and the term may as well be dropped. When the two fluids, at different potentials, attempt to become equalized, they pass along certain paths and form *electrical currents*. Elec-

tricity will pass along any substance, but some substances conduct it more easily than others, and these are known as *conductors*. The relative value of different conductors is shown in the following table:

1. The metals; 2, charcoal; 3, plumbago; 4, dilute acids; 5, saline solutions; 6, pure water; 7, living animals; 8, flame. Of the metals, silver and copper are the best conductors. The human body would have about the same conductibility as the saline solutions, if it were not for the skin, which is a very poor conductor, especially when it is dry. Those substances along which electricity passes with great difficulty are known as non-conductors or *insulators*. The following is a list of some of these, the substances arranged in accordance with their relative value:

1. Caoutchouc. 2. Silk. 3. Glass. 4. Wax. 5. Sulphur. 6. Resins. 7. Shellac. 8. Dry air.

The electrical fluids may be kept by insulators at different potentials, the insulators preventing them from becoming equalized or reduced to the same potential. Electricity in this condition is called static. Its study is called electrostatics.

The electric fluid in motion is called dynamical electricity, and its study is electrodynamics.

Technical Terms.—There are certain technical terms which it is necessary to understand. *Electromotive force* (symbol, EMF) is the force which tends to set electricity in motion. An electric current results. The *current strength* (symbol, C) is the term used to express the capacity of the separated fluids to overcome resistance in their attempts to reach equilibrium or equalization again. This current strength, or simply the current, naturally, is in proportion to the strength of the electromotive force, which is constantly dissociating the electrical fluids and generating the current. If, however, as is always the case, the electrical fluid meets resistance in seeking equilibrium, the resistance diminishes its current. Hence we have the formula known as Ohm's law:

$$\text{Current strength} = \frac{\text{Electromotive force}}{\text{Resistance}}; \text{ or } C = \frac{EMF}{R}.$$

All bodies offer some resistance to electrical currents, and it is important to have some standard unit of resistance for the sake of comparison. Such standard unit has been adopted and is called an *ohm*. It is the resistance offered to a current by a certain piece of wire of definite size and length.

A *volt* is the unit of electromotive force, *i.e.*, it represents the force which will generate a certain amount of electricity in a second of time. A Daniell cell is of not quite one volt strength.

An *ampere* is the unit of working power or current strength. It

is the current strength produced by one volt of electromotive force working against one ohm of resistance. A milliampere is one-thousandth of an ampere. A *watt* is the unit of work.

When a given current flows along from a large into a small conductor, the quantity in this latter conductor in a given section is greater and the current is said to be denser. The instrument by which the strength of a current is measured is known as the amperemeter: in medical practice, only fractions of the ampere are used, and the instrument is called the *milliamperemeter*. A *rheostat* is an instrument for interposing resistance in a current.

Electrical Appliances.—The batteries used in neurological practice are of three kinds: the static, the faradic, and the galvanic.

The static electrical batteries are mostly modifications of the Holtz influence machine. They are inclosed in glass to prevent the effects of moisture. The instruments made in this country for medical purposes will usually furnish electricity all the year round. The battery accessories consist of an insulated stool and brass-ball electrodes with glass handles. The patient is placed on the stool, which is connected by a rod with one of the prime conductors. The battery being started, the patient becomes enveloped, as it were, with a layer of electricity which is at a very high potential and constantly flying off, being retained only by the dry, non-conducting air. The electrode held by the operator, and connected directly or indirectly to the other prime conductor, is now brought near the patient. The electrical fluid bounds to the zero point with such force that some of the metallic parts of the electrode are carried off and ignited, causing the disruptive spark. The patient thus is discharged of the fluid; but it continually reaccumulates upon him, and thus one can keep on drawing sparks from all parts of the body. The electric spark causes a muscular contraction and, a little later, a small punctate red spot. There is some pain connected with it, but as the electricity penetrates the body for only an infinitesimal period of time, it directly affects the viscera but little and is not dangerous. Various ingenious electrodes have been devised for modifying the character of the static discharge, but they all have much the same effect as the spark.

By approximating the prime conductors so that they almost touch, and then connecting the outer surface of the two Leyden jars which hang from the prime conductors with sponge electrodes, one gets the static induced current described by Dr. William J. Morton. The special peculiarity of this current, as well as of the spark discharge, is that it is made up of electricity at a very high potential, and that, being composed of oscillating or alternating currents of extremely short duration, the quantity of electricity is very

small. The result is that when a muscle degenerates, it loses its irritability to the static current very early. The static current will thus reveal a beginning degeneration of nerve sooner than the other currents; it also promotes tissue changes more than other forms. It sometimes stimulates a greatly degenerated muscle, so that later the other forms will produce a contraction. It has a powerful psychical effect, and lends itself readily to quackery.

In *faradic medical batteries* the electricity is produced by induction. The chief elements are a cell and two coils of insulated copper wire. One coil is placed around the other, the outer coil being longer and of finer wire. The cell generates a current which in turn "induces" the electricity which is received by the patient. The original current is so arranged that it is being constantly broken and closed or "made" again. At both break and make, a current is induced in the inner coil of coiled wire; this forms what is called the *primary induced current* and is made up of a succession of short currents. This current, though theoretically alternating, is really a current of one direction. For the "make" current is opposed and nullified by the original or battery current. The same is true of the *secondary induced current* which is excited in the outer coil by the currents of the inner coil. Both currents are really composed of a rapid series of single currents going the same way, and they each have a positive and a negative pole. The primary current is one of lower tension, and is rather less strong. It can be used when the resistance of the parts is not great and a very powerful current is not needed. The secondary current is one of higher tension, it overcomes resistance better, and can be employed in connection with the wire brush and in anæsthetic conditions. It is also the current used in measuring the strength of the faradic application, as will be shown later. The current of the secondary coil resembles that of the static induced current. It has, however, a lower potential, vastly slower alternations, and more quantity. It can contract muscle, which the static current cannot affect. The secondary coil should be made of wire of a standard size, length, and layers of coil. A standard coil of wire 800 metres long, .225 mm. in diameter, wound on a spool 100 mm. long, is quite generally adopted now.

Faradic batteries are made with the zinc-carbon or Grenet cell, the Leclanché, the silver-chloride cell, or some modification of these. For general use, the zinc-carbon cell is the most trustworthy; but the dry silver-chloride cell is the most convenient. A dry cell made with muriate of ammonia has also come into use. Faradic and galvanic currents are now often obtained from the electric-light wire now so generally supplied to houses.

Galvanic Batteries.—There are two kinds of these in practical

use. The one includes the cells which act as soon as the circuit is closed; the other includes those which act only when the elements composing the battery (*e.g.*, zinc, carbon, platinum, copper, etc.) are dropped in the exciting fluid. The former class (known as two-fluid cells) are not touched except to renew water or add some chemical. In the latter (the single-fluid cells), one of the elements is always taken from the fluid when the current is not in use. The first class of cells has a much weaker chemical action and evolves less electricity in a given time. It includes the Daniell cell, the gravity cell, the Leclanché cell, and the silver-chloride cell. Among the second class or single-fluid cells, the zinc-carbon cell, known as the Grenet or Stohrer's cell, already referred to, is most used. The best portable batteries are made of the zinc-carbon cell

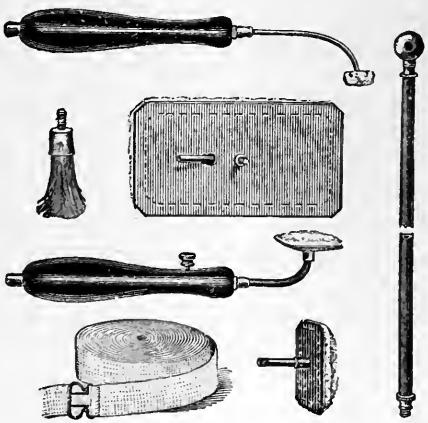


FIG. 35.—AUTHOR'S ELECTRODE SET.

or the dry chloride of silver cells. Stationary or office batteries are best made with the Leclanché cell or some modification of it; or the dry silver-chloride cell may be used. The electric-light current can be utilized to supply continuous and interrupted currents and for purposes of the cautery. It is expensive.

The ordinary accessories to the faradic and galvanic batteries are electrodes, rheostat, and milliamperemeter.

The electrodes needed for ordinary purposes are: An indifferent electrode measuring 5 cm. by 15 cm. A normal electrode, 10 sq. cm. A unit electrode, 1 sq. cm. A soft wire brush. Three handles: one 10 cm. and one 40 cm. long, one short handle with an interrupter. A milliamperemeter. A rheostat. (See Fig. 35.)

Methods of Application.—Static electricity is applied for fifteen or twenty minutes daily or tri-weekly. For general tonic or sedative effects, sparks are drawn from all parts of the body except the face. In paralysis or spasm or pain, sparks are applied to the

affected area. For headaches and cerebral paræsthesiæ, the electrical breeze is very useful, but it must be strong.

The faradic and galvanic currents are used for about the same time and intervals as the static. In some cases, however, the galvanic current should be given daily or even two or three times a day. As a rule, a course of electrical treatment should be continued for six to eight weeks, and then discontinued for a time.

The special methods used in applying these currents are:

1. General galvanization and faradization or general electrization.

2. Local electrization by galvanization of the brain, of the neck, of the spine, of the special senses, limbs, and viscera. Or by faradization of the neck, spine, limbs, and viscera.

3. The combined faradic and galvanic currents. These are given by means of the De Watteville switch. General and local electrization can be given in this way.

4. The polar method. This is employed chiefly in using the galvanic current. The indifferent electrode is placed on the sternum or back, and the other electrode applied wherever indicated.

5. Cataphoric electrization by means of Peterson's electrode.

6. Electrolytic applications are used in enlarging strictures and affecting inflammatory deposits and neoplasms.

In general electrization, whether galvanic or faradic, the indifferent electrode is placed on the sternum, feet, or back, and the other pole is carried over the limbs, trunk, neck, and, if indicated, the head. In some cases, however, the two electrodes are applied together upon the different muscles of the body. In local electrization, the large electrode may be applied on an indifferent spot, and the other applied to the affected limb or limbs, or the two electrodes may be used together on the same segment of muscles. There are special points at which the muscular contraction is most easily brought out. These are called the motor points. See Figs. 36 to 41. In the ordinary practice of applying electricity for spinal-cord disease, with galvanic currents, a very minute amount of electricity reaches the cord. With large electrodes, however, and currents of 50 to 140 milliamperes the cord is reached with a current of $\frac{1}{2}$ to $1\frac{1}{2}$ ma. strength. In locomotor ataxia one (positive) electrode, six by twelve inches, is placed on the upper part of the back, and a second electrode of the same or larger size placed on the abdomen, the lowest part of the back, the legs, and the perineum. The current should be increased very gradually and should be kept on for only a minute in each place. The method must be varied somewhat according to the size and sensitiveness of the patient. The details

for galvanizing the brain, special senses, and viscera must be obtained from special text-books.

When an electrode is held steadily upon a part, it is called

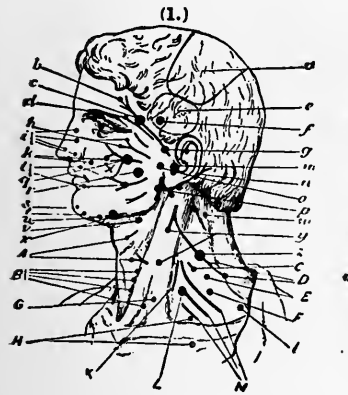


Fig. 36.

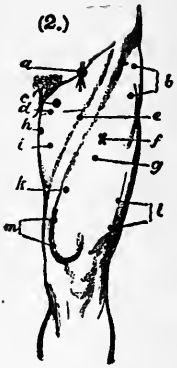


Fig. 37.

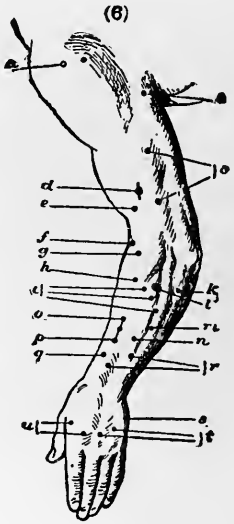


Fig. 41.

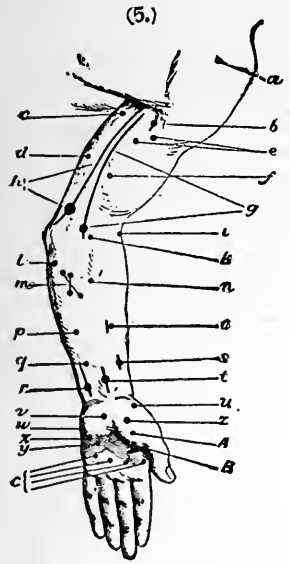


Fig. 40.

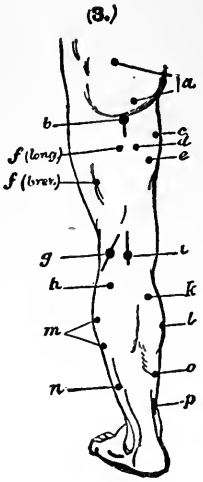


Fig. 38.

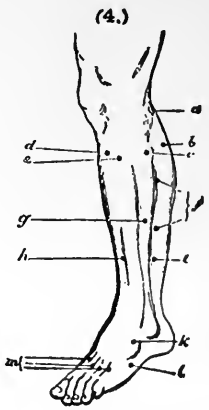


Fig. 39.

Fig. 36 to 41.—Cuts Showing Motor Points. The letters refer to points electrization of which stimulates certain muscles to contract. These points vary much in different persons. For details see works on Medical Electricity.

stabile; when it is moved over it, it is called *labile.* The positive pole is called the anode (An), the negative the cathode (Ca). The size of an electrode is indicated in square centimetres.

A formula for applying electricity may be written thus:

Anod. galvanization, 10 ma. 5×15 cm. daily, 5′ stabile.

This means that the positive pole of the galvanic current is to be applied steadily at a given place daily for five minutes with an electrode of 75 square centimetres.

The terms "ascending" and "descending currents" are rarely used, the name of the pole being employed instead. Thus, anodal galvanization of the brain or arm means that the positive pole is applied at these localities. With the faradic and static currents, neither the pole nor the direction of the current makes much difference.

Electro-Diagnosis.—When a motor nerve is cut off from its centre in the spinal cord, or when this centre itself is diseased, the nerve and later the muscle undergo a degeneration. As a result of this, their reaction to electrical currents is changed, and we get what is termed "partial degeneration reactions" and "complete degeneration reactions," according to the degree of disturbance. These reactions are due mainly, if not wholly, to the degeneration in the terminal nerve fibres and motor end plates in the muscle. When the muscle alone is diseased, the reaction is not changed until very late. The change in irritability is due to the fact that as the nerve fibre wastes it takes an electric current of comparatively long duration and considerable strength to stimulate it.

The first effect is to cause it to lose its contractility or reaction to weak currents, then to extremely rapid, short currents like the static, then to the faradic, and last to the galvanic. Such change is known as the *quantitative alteration* in electric irritability.

But besides this, the nerve and muscle are affected in a different way by the different poles of the galvanic battery. In normal nerve and muscle, a contraction is caused more readily by the negative pole than by the positive. But muscles with degenerated nerve supply sometimes respond as well or better to the positive pole. This forms what is called the *qualitative* or *serial change* in the irritability of the muscle.

Finally, degenerated muscles respond more sluggishly than normal to the galvanic and faradic currents. The contraction, instead of being sharp and jerky, is sluggish and almost tetanic. This is called the *modal change* in irritability, and it is far the most important sign of muscular degeneration.

The *qualitative* change is gotten only by placing the active electrode over the muscle, but the *quantitative* and *modal* changes may be gotten by placing the electrode over the nerve as well as over the muscle. In describing these changes, the following abbreviations are used:

DeR = degeneration reaction.
AnCC = anode or positive-pole closure contraction.
CaCC = cathode or negative-pole closure contraction.
AnOC = anode opening contraction.
CaOC = cathode opening contraction.
Te = tetanus.
D = circuit is closed and current flowing.
AnDTe = tetanic contraction while the positive pole is applied and the circuit closed.
The sign > means greater than; <, less than. Thus AnCC>

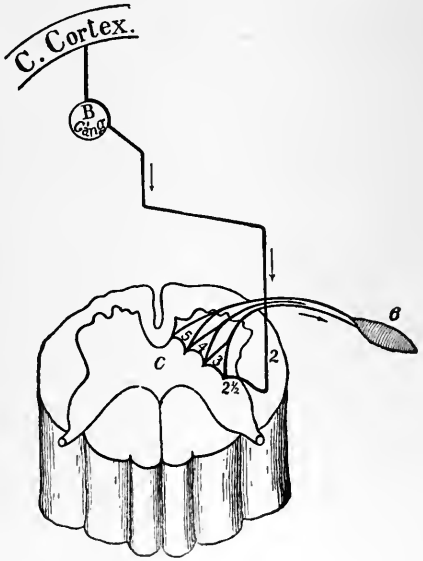


FIG. 42.

CaCC means anode closure contraction is greater than cathode closure contraction.

Degenerations in nerve do not occur except in lesions of the nerve or spinal cord, and in very late stages of primary atrophy of muscles. Hence when one finds degenerative reactions, he can almost absolutely exclude disease of the brain, functional disease, and primary disease of the muscle. The following rules may be formulated for testing for degeneration reactions:

Use the faradic current first.

The Faradic Current.—Use a secondary induction coil of wire .225 mm. in diameter and 800 metres long. The distance over which the coil moves is divided into a hundred parts. The strength of current is indicated by percentage or millimetres. In many scales it takes 30 to 40 mm. of CD or coil distance to cause a muscular contraction. Record the minimum necessary for muscular contrac-

tion, using the same electrodes and in the same way as in testing with galvanism.

The Galvanic Current.—1. Place the indifferent pole over the sternum, and a 10 sq. cm. electrode over the muscle. 2. Pass the current for one minute. 3. Then find the minimum strength needed for a cathode closure contraction. 4. Then for an anode closure contraction. Repeat this test three times. 5. With a given current, note whether the cathode closure contraction is stronger than AnCC, or otherwise. Test this three times. 6. Note the character of the contraction, if sharp or sluggish. 7. Test nerve in same way.

The qualitative changes may be expressed by a formula like AnCC = or > CaCC, *i.e.*, the positive-pole closure contraction is equal to or greater than the negative-pole closure contraction. Or, better, the minimum strength of current required to cause a contraction in the muscle is recorded for the positive pole and for the negative. Thus:

AnCC 5 ma. or 8 cells.
CaCC 4 ma. or 6 cells.

The following table (modified from De Watteville) and diagram (Fig. 42) show the diseases in which degeneration reactions may be expected:

TABLE SHOWING THE LESION, ITS RESULTS, THE NAMES OF THE DISEASES, AND THE ELECTRICAL REACTIONS.

Lesion of—	Result.	Disease.	Electrical Reaction as to Qualitative.
1 to 2½. Cortex to cord.	Paralysis, contractures.	Hemiplegia from hemorrhage. Embolism, tumors, lateral sclerosis.	Nerve: normal. Muscle: normal.
3, 4, and 5. Coruua.	Paralysis, degenerative atrophy of nerve and muscle.	Acute and chronic anterior poliomyelitis.	Nerve: DeR. Muscle: DeR.
2 to 2½. 3 to 5. Lat. cols. and ant. corn.	Paralysis, contractures. Degenerative atrophy of muscle.	Amyotrophic lateral sclerosis.	Nerve: normal / Muscle: partial } DeR.
5 to 6. Trophic cord centres.	Degenerative atrophy of muscle. Paralysis from wasting of muscle. Later, degeneration of nerve.	Progressive muscular atrophy (spinal form), bulbar paralysis (?).	Nerve: normal. Muscle: or partial DeR. When disease is advanced.
Nerve.....	Paralysis; degenerative atrophy of nerve and muscle.	Neuritis; from wounds, toxæmia, or pressure.	Muscle: DeR. Nerve: DeR.
Muscle	Wasting, paresis.	Simple atrophy; primary or idiopathic myositis. Juvenile form of progressive muscular atrophy; pseudo-muscular hypertrophy; other types of primary myopathies.	Nerve and muscle normal until late in the disease.
Nerve and muscle.	Paresis and atrophy.	Rheumatic atrophy and paresis.	No DeR. unless severe.

It should be said, finally, that it is the *sluggish contraction* which is the most important element in showing degeneration: also that it is the muscle which should be tested most carefully, as only over it does one get the qualitative changes.

Therapeutics.—Electricity is used as a counter-irritant and as a general mechanical tonic in states of muscular and nervous weakness. It is used in paralysis, spasm, and pain, and for its supposed specific action in certain functional and organic diseases.

The faradic and static currents have a counter-irritating, stimulating, and excito-reflex effect. The galvanic current has a sedative and antispasmodic effect.

Electrolytic, cauterizing, and cataphoric effects are also produced, but are rarely needed by the neurologist. A considerable portion of the effects of electricity are psychical, but they are not the less real or valuable.

CHAPTER VII.

GENERAL DISEASES OF THE PERIPHERAL NERVES.

Introduction—Anatomical.—The peripheral nervous system consists of twelve pair of cranial and thirty-one pair of spinal nerves with their root ganglia and terminal sense organs, and the sympathetic nervous system. The sympathetic system is composed of the intervertebral and the cranial ganglia, and the peripheral ganglia. The latter arise during embryonal life from ganglionic cells of the same class as those of the spinal root ganglia, and migrate to their adult position in the sympathetic (Minot). The peripheral nervous system, therefore, to use the language of modern anatomy, is composed of peripheral motor neurons, peripheral sensory neurons, and peripheral ganglionic neurons (Minot).

The Origin of Nerves.—The recent discoveries of the true nature of the nerve cell and its relation to the nerve fibre have made a great difference in our conception of the nervous centres. We now know that the peripheral nerves are really only the prolongations of the axis-cylinder processes of the nerve cells, with certain anatomical additions which nature has made in order to isolate and support them. The peripheral nerve fibres are only a part of certain neurons. The motor nerve fibres come from the motor nerve cells, and form the peripheral part of the peripheral motor neurons. The sensory fibres are derived from the sensory nerve cells, and are only a part of the sensory neurons. All peripheral nerve fibres of motor nerves have, in the spinal cord or brain, certain cells of origin, which are known as the *nuclei of origin* of these nerves. These motor nuclei lie in the anterior and lateral horns of the spinal cord, and in the corresponding parts of the medulla and pons. It has been customary to refer the sensory nerve fibres also to *nuclei of origin* in these same parts. This, however, according to modern views, is not correct. All sensory nerve fibres take their origin from nerve cells in the posterior nerve ganglia, or in corresponding cranial ganglia, such as the petrous and the jugular, lying upon the roots of the cranial nerves. The nerve cells in these ganglia send off a single process which divides in a T-shaped fashion, the peripheral branch going out to form the sensory fibre, the central branch passing into the cord or brain, to end in a terminal arborization which surrounds groups

of sensory nerve cells. These sensory nerve cells in the cord are, therefore, not really nuclei of origin, but are terminal nuclei. There are no nuclei of origin for sensory nerves in the central nervous system. This changed point of view is of especial importance in our consideration of the anatomy of the cranial nerves (Fig. 43).

General Pathology.

Hyperæmia and Anæmia.—Under the head of hyperæmia and anæmia there occur types of nerve irritation, leading to different forms of neuralgia, paræsthesia, and motor weakness or irritation. Hyperæmia and anæmia are, however, secondary conditions and are rarely recognized clinically. It cannot always be determined

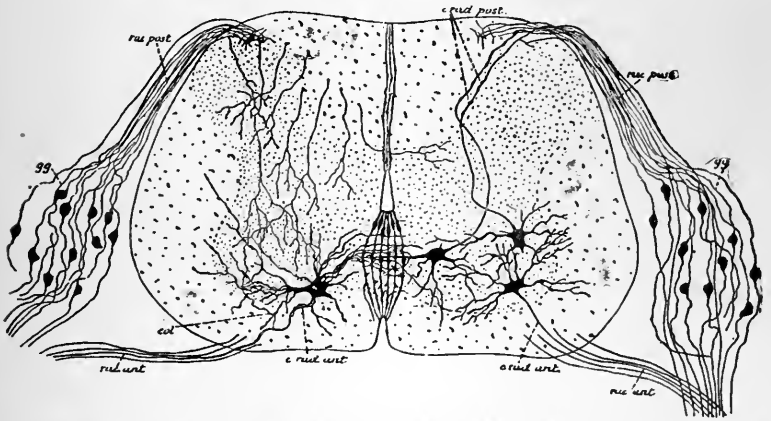


Fig. 43.—Showing the Cells of Origin of the Motor Nerves in the Anterior Horns of the Spinal Cord, and the Cells of Origin of the Sensory Nerves in the Posterior Spinal Ganglia (Van Gehuchten).

whether an irritated nerve is congested or anæmic, or whether the central part of the nervous system is not mainly at fault.

Inflammation of Nerves—Neuritis.—There are two forms of neuritis: 1. Interstitial neuritis and perineuritis. 2. Diffuse neuritis with parenchymatous degeneration. The first form may be acute or chronic.

In the first type there is hyperæmia, with sometimes extravasation of blood. An exudation occurs into the fibrous framework of the nerve, with migration of leucocytes. The inflammation may become suppurative or gangrenous. If severe, it destroys the nerve fibres; but oftenest the axis cylinders are not destroyed, and recovery takes place. Chronic interstitial neuritis and perineuritis are accompanied with hyperplasia of the connective tissue, compression and more or less destruction of the nerve (Fig. 44). It may

ascend or descend, and it is called, accordingly, *ascending, descending,* or *migrating* neuritis. It may affect only certain segments of the nerve, when it is called *segmental* neuritis or disseminated neuritis. Tuberculous and syphilitic neuritis are of the chronic interstitial or diffuse type. These latter forms rarely involve peripheral nerves, but rather the intracranial parts of the cranial nerves and the spinal nerve roots in meningeal tuberculosis or syphilis. A syphilitic peripheral multiple neuritis is, however, thought to occur sometimes. Leprous neuritis is a very typical form of proliferating

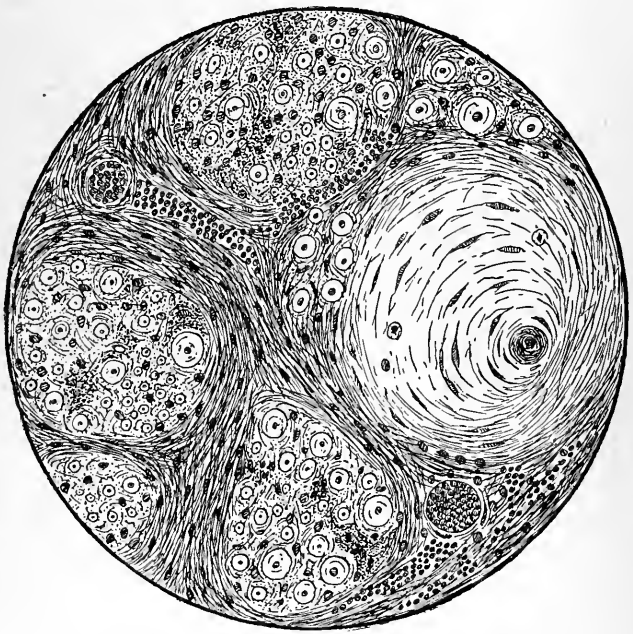


FIG. 44.—ACUTE INFECTIOUS NEURITIS, showing hemorrhage, connective-tissue proliferation, diseased nerve fibres, and obliterated vessel (Rosenheim).

chronic perineuritis. Cancerous neuritis sometimes occurs, and it is of the diffuse type, though sometimes an actual cancerous process invades the nerve.

The second type is called degenerative neuritis and this process of degeneration is the dominant one, so that the changes can be best described under the head of degeneration of nerves:

DEGENERATION OF NERVES.—This is a process in which the nerve fibres gradually die; the myelin sheath and axis cylinder disappear, leaving only a strand of connective tissue.

Nerve Degeneration.—There are three forms of nerve degeneration: 1. Primary; 2. Secondary; 3. Neuritic or toxic.

1. The primary form is rare, slight in extent, and of little clinical significance. In it there is simply a gradual wasting and disappearance of the axis cylinder and myelin sheath. It occurs in old age, wasting diseases, and as part of locomotor ataxia (Fig. 45).

2. Secondary degeneration or Wallerian degeneration. This form occurs when the nerve is cut across, or compressed, or destroyed by inflammation, neoplasms, or injuries.

The essential part of the nerve fibre, the axis cylinder, is simply a prolongation of the process of a nerve cell. Its next essential part is the myelin sheath. This is of epiblastic origin and consists of a hollow cylinder inclosed in a thin membrane and containing a fatty substance. In degenerative processes of peripheral nerves the medullary sheath is first affected, then the axis cylinder, least and

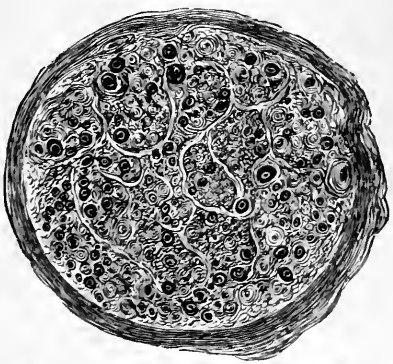


FIG. 45.—SIMPLE ATROPHY OF NERVE IN MARASMUS.

last the neurilemma. The myelin becomes turbid, splits up into fragments and droplets. The axis cylinder also breaks up into fragments or swells up and becomes liquefied. Extravasated leucocytes pick up the products of disintegration and form fat-granule cells. The neurilemma and its nuclei usually remain intact. The nerve during this time shrinks in volume and looks grayish and translucent, or grayish-red. The nerve finally become only a fibrous cord. Changes can be seen in the nerve within forty-eight hours, and by this time its irritability, which was first slightly increased, is lost. In about two weeks the disintegration of the myelin sheath and axis is practically complete (Fig. 46). The peripheral end of the cut nerve shows a loss of nearly but not quite all the fibres as far as its termination. In the central end, the degeneration ascends at first only to the first or second node of Ranvier. Very soon, however, a change occurs in the cell from which the fibre springs. This change is called the *reaction at a distance*, or degeneration of

Nissl. Thus when the neuraxon is injured the whole neuron suffers, but the peripheral end far the most.

When a section is made between the spinal ganglia and the cord, the fibres all degenerate toward the cord, and even within it, but the peripheral fibres do not degenerate. Hence the spinal ganglia are the trophic centres of the sensory nerves (see Fig. 47).

Degeneration occurs in the motor nerves, also, when the cells of the anterior horns are destroyed. Hence these cells are the trophic centres for all motor nerves. The process of degeneration takes place at about the same time throughout the whole length of the nerve. The motor end plates in the muscles are affected a little the earliest. About the cut end, little bulbous tumors may de-

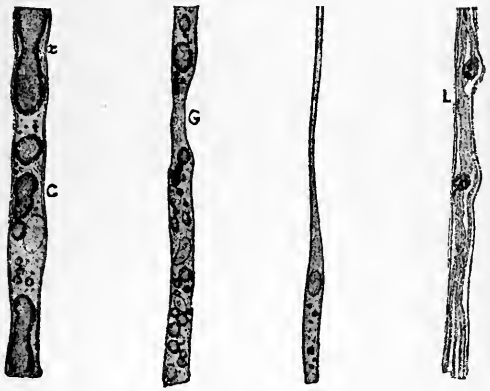


FIG. 46.—SHOWING DIFFERENT STAGES IN THE PROCESS OF NERVE DEGENERATION ON SECOND, THIRD, SIXTH, AND NINETIETH DAYS AFTER SECTION (Ranvier).

velop, which contain numerous nerve fibrils and connective tissue. The general law is that nerves degenerate in the direction in which they carry impulses, but this is not the whole case, as has been just described. If the injury to the nerve is permanent, a slow decay affects the whole neuron. Supposing for example a motor nerve is injured or inflamed at the point *D*. Immediately a degeneration takes place along the parts below to *T*, and in a few days a slight degeneration takes place in the cell body *C* (see Fig. 48).

Summary: Peripheral nerve fibres degenerate when cut off from their trophic cells. The degeneration begins at once throughout the length of the nerve. Loss of function occurs in forty-eight hours. The degeneration is practically complete within two or three weeks. The myelin sheath and its nuclei are affected first, the axis cylinder next. The degeneration takes place most quickly and markedly in the direction in which the nerve impulse runs, except in peripheral afferent nerves. The central end of the nerve

and its cells of origin undergo a slower and milder degeneration. The final stage is one of nerve atrophy or of nerve degeneration.

Within *the central nervous system* degeneration also occurs mainly in the direction of the nerve impulse. The axis cylinder is first affected. There is sometimes a preliminary swelling or hypertrophy of this axis cylinder. Degeneration with calcification of the nerve

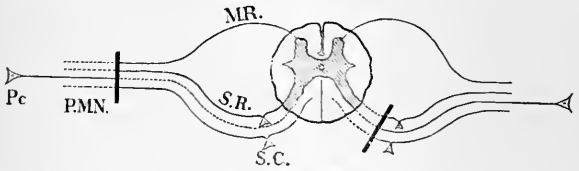


FIG. 47.—SHOWING EFFECTS OF SECTION OF MIXED NERVES AND SENSORY ROOT. *P.MN.*, Section through mixed nerve; *MR.*, motor root; *S.R.*, sensory root; *S.G.*, spinal ganglion.

fibres sometimes occurs. In associative or commissural fibres the degeneration extends only part of the length of the nerve.

3. Neuritic or toxic nerve degeneration. This form occurs in connection with neuritis, and will be described under that head. Its chief characteristics are that the degeneration attacks the nerve in segments, that the axis cylinders are not so much affected, and the myelin breaks up into small fatty droplets instead of into large masses. The same general laws apply to it as to Wallerian degeneration.

Degenerative processes in the non-medullated nervous fibres have been observed in the fine fibres of the cornea and in the submucous and myenteric plexuses of the alimentary tract.

Regeneration of nerves is a process that usually follows degeneration. It occurs only in peripheral nerves—very little, if at all,

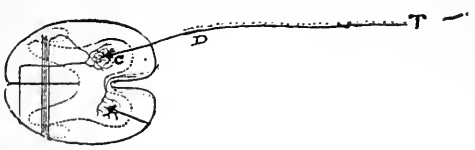


FIG. 48.

in the nerves of the central nervous system of mammals. It is an unique process, in that the nerve is the only specialized tissue that can grow again after being destroyed. Regeneration occurs whenever the trophic centres are healthy, when the mechanical obstacles to a union of the divided fibre are not too great, and when the peripheral nerve is not too completely atrophied. It occurs most quickly, therefore, when the cut ends are carefully apposed and when the separation has not lasted for a long time, *i.e.*, for years.

It progresses always from the *central* end toward the periphery. The fibres of the central stump grow out into the degenerated peripheral fibre. Union by *first intention* or *second intention* never occurs. Human nerves cannot be made to unite physiologically, but only anatomically.

Regeneration may be complete in a few months in short nerves. In the sciatic it may take one or two years. When regeneration takes place, the axis cylinders of the central stump swell and divide into a number of new cylinders which pierce or creep around the intervening tissue, enter in bundles the peripheral nerve, and become inclosed in new myelin sheaths and neurilemma.

General Symptoms.

One of the commonest forms of symptoms occurring in the distribution of the peripheral nerves is paralysis and atrophy of muscles in a greater or less degree. But since the motor nerve carries with it some vasomotor and secretory fibres, there may

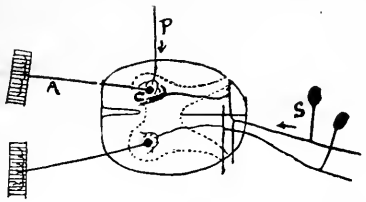


Fig. 49.

also be congestion, œdema, sweating, etc. When the motor neuron is damaged only to a moderate extent or is irritated by any agent, twitchings and spasmodic movements of the muscle may occur. Spasmodic symptoms in the distribution of peripheral nerves may also be due to disturbances acting upon the central motor cell. The seat of the trouble is either in the sensory neuron, as in reflex spasmodic troubles, or in higher motor neurons. Thus, in a spasmodic disorder affecting the motor nerve *A*, the irritation may be in the cell body *C*, but is more often reflex, starting in the sensory neuron *S* or in a higher motor neuron whose neuraxon is represented at *P* (Fig. 49).

A symptom that is also very common in disease of the peripheral nerves is neuralgia, or the milder degree of nerve irritation called paræsthesia. We have besides this various forms of anæsthesia, which is due to cutting off the sensory fibre wholly. Neuralgia in its simpler and commoner pathological forms is due to some irritation of the peripheral sensory neuron, especially its ganglionic centre. The particular nerve affected tells the location of the irri-

tation. But the peripheral sensory neuron is in close relation with the central neurons that carry the painful impressions to the brain. Hence the whole sensory path may become disordered, and this is particularly the case in young and neurotic patients. As a consequence we find that neuralgias and pains are more diffuse and less sharply localized at this period of life.—Besides this, it sometimes happens that the central sensory neurons in the brain are themselves the seat of injury or irritation, and we have at times, therefore, central neuralgias. However, there can be no doubt that the central sensory neurons are not easily irritated to the point of exciting pain referred to a peripheral nerve, and this is because the ascending sensory tract is a widely diffused one and not massed in a single compact fasciculus.

It will be seen from the foregoing that the nervous diseases affecting the functions of the peripheral nerves will fall into three classes: first, those affecting a large part of the spinal nerves, at the same time causing general symptoms of paralysis, pain, etc.; next, diseases involving especially certain nerves of motion; and finally, those affecting especially nerves of sensation. So I shall describe general neuroses involving the functions of many and mixed nerves, then neuroses in the limits of the motor, and those in the distribution of sensory nerves.

MULTIPLE NEURITIS—POLYNEURITIS.

Originally multiple neuritis was described as though it were always the same disease. Later investigations show that this is not a true view to take of it because nerves are inflamed in very different ways and degrees, and because the causes of the inflammation affect the general system so differently. A diagnosis of multiple neuritis alone is, therefore, not sufficient. The chief recognizable forms of multiple neuritis are: 1. The motor form, or paralytic neuritis. 2. The sensory or ataxic form of neuritis (pseudotabes). 3. Endemic neuritis (beriberi). 4. Acute pernicious neuritis (Landry's paralysis).*

The Motor Type of Multiple Neuritis (*Alcoholic Paralysis, Arsenical Paralysis, Diphtheritic Paralysis*).—This is the common form of the disease, making up fully ninety per cent of all

* Among 71 cases of multiple neuritis of which I have analyzed the notes there were of the common motor form, 62; sensory or pseudotabetic, 4; endemic (beriberi), 3; acute pernicious, 2.

Over half of the motor type were confessedly alcoholic, 34. Of the others, there were: Post-diphtheritic, 8; measles, 1; grippe, 2; erysipelas, 1; sepsis,

cases seen in this country. While sensory and other symptoms always accompany this type, it is the paralysis and atrophy of muscles which are the leading and chronic conditions. It is a malady coming on acutely, running a subacute or chronic course, rarely fatal, and characterized by weakness or paralysis of all four extremities associated with atrophy, pain, tenderness, and various vasomotor, secretory, and trophic disturbances. It is a disease pre-eminently due to poisons and infections, alcohol being far the commonest factor.

It occurs oftener in the female, owing to the fact that alcohol poisons the nerves of women more than of men, and perhaps because of the predisposing influence of tea-drinking.

Multiple neuritis is essentially a disease of early adult life. Almost all cases occur between adolescence and the period of degenerative changes, twenty to forty-five. Young children are very rarely subject to it, but cases have been reported occurring in children at the age of seven, nine, ten, and fourteen, and G. M. Hammond has reported several cases occurring in infants. A few cases have occurred in persons over sixty; but they are only a little less susceptible than children.

The sporadic forms of polyneuritis from alcohol and various poisons and infections occur without much reference to a seasonal influence. Probably more cases occur in spring and fall, owing to sudden changes in temperature. Epidemic influences like that causing cerebro-spinal meningitis may increase the number of cases of multiple neuritis. Practically, in this country, the question of drink settles the question of the distribution of polyneuritis. It is rare in the temperate rural districts and smaller towns, and much rarer in native Americans than in foreigners.

Sexual excesses, exposure to cold and wet, insufficient diet, excessive tea-drinking, the presence of tuberculosis, predispose to the disease. The same neuropathic tendency leading persons to excesses in alcohol, tea, and to suicidal indulgence in arsenic is of some moment in leading to the development of neuritis.

The list of the special and exciting causes is long and includes nearly all infectious fevers, many chemical and autochthonous poisons. The common infections are diphtheria, puerperal and

1; puerperium, 4; diabetes, 2; metallic poison (lead), 2; ptomaine poisoning, 1.

The sex of 44 was male, 27 female, but in the alcoholic cases the sex was female in the proportion of 10 to 1.

The age ranged from under 10 (diphtheritic cases) to 67. Most cases occurred between 31 and 40 (25 cases), next between 21 and 30 (18), and next between 41 and 50 (15).

other septic fevers, and endemic infections of unknown origin. Nearly every infectious fever and malaria may be added to the list.

Of chemical poisons alcohol heads the list, causing over two-thirds of the adult cases. Next come arsenic, lead and phosphorus, and copper. Among the autochthonous poisons, rheumatism, diabetes, and the metabolic products resulting from starvation and cachexia lead to multiple neuritis.*

Fig. 50.—Alcoholic Paralysis, with Foot Drop and Wrist Drop.

Among the foregoing causes, arsenic and diabetes produce more often decided sensory symptoms.

Prodromata. — The disease often begins with prodromata lasting several weeks. The patient suffers from numbness, slight pains, and weakness affecting especially the lower extremities. Sometimes a peculiar condition of mental confusion and weakness precedes the attack. Usually the symptoms come on rather suddenly. The patient suffers from pains and tenderness in the legs and feet, and is obliged to go to bed. There may be a fever for a day or two, the temperature rising to 102° or even 104° F., but this is not the rule. The pains and weakness increase. The muscles and nerves are very tender. The fingers, hands, and arms are often similarly but less affected. At the same time the skin becomes reddened or slightly œdematous. The muscles of the legs grow weak, and in a day or two the patient is unable to stand. In a week or two there may be a complete loss of power in the anterior tibial muscles and a lesser degree of paralysis in the extensors of the hand (Fig. 50). Nearly all of the leg and forearm muscles become eventually involved. Atrophy sets in at the same time and very severe pains are present. The motor cranial nerves are in rare cases affected, and paralysis of the facial or of the third, fourth, or sixth nerve has been seen. When the disease is fully developed, which is within a fortnight, there is paraplegia.

* Trional is to be added to the list of drugs which may cause neuritis.

with foot drop, some degree of wrist drop, muscular atrophy, and slight œdema, especially of the feet. The skin reflexes are often, the knee jerk and elbow jerk always lost. There is some tactile anæsthesia, often with hyperalgesia. Temperature and pain sense are also lessened and slowed in transmission. The anæsthesia occurs in patches or diffusely. Muscle and articular sense are lost in the sensory or pseudo-tabetic form, and are usually somewhat involved in the ordinary paralytic form. Pain and sensitiveness continue.

The nerves lose their irritability and the muscles show degeneration reaction, partial or complete, the characteristic being a great variability of reaction over different groups of nerves and at different stages of the disease, and an early loss of faradic and lessening of galvanic irritability. There is sometimes retinal hyperæmia and even optic neuritis. Of the visceral nerves, the vagus seems oftenest to show signs of involvement, in rapid pulse and disturbances of respiration. The sphincters are rarely involved and then only for a few days. In such cases there is, perhaps, involvement of the cord or of the abdominal and pelvic splanchnics. In alcoholic and occasionally in other forms of neuritis, mental symptoms, such as a low, muttering delirium, are very often present, and occasionally a well-marked confusional insanity develops.

The disease usually reaches its height in a week or two and then starts on a chronic course; but it sometimes happens that exacerbations occur, or that a paralysis and atrophy progress for several weeks before regression begins. In alcoholic cases there is often great general prostration; the patients lie for several weeks in a delirious condition, and finally develop pneumonia and die.

In *diphtheritic neuritis* some of the eye and throat muscles are involved, while the extremities are usually but slightly or temporarily affected and the sensory symptoms are few.

Some further details should be added.

Motor Symptoms.—The characteristic paralysis of multiple neuritis is a quadruplegia, all four extremities being involved. The special characteristic is the foot drop, which is indicative of alcoholic neuritis, just as wrist drop is of lead palsy. The paralysis is typically a peripheral one. It affects the feet and legs, hands and forearms. It usually involves the anterior tibial muscles more than the calf muscles, but sometimes the reverse occurs. The muscles become wasted and flabby. They soon lose in bad cases all reaction to faradism, and they require a strong galvanic current to produce a contraction. In anterior poliomyelitis, on the other hand, the diminution in galvanic irritability comes on only after weeks or months. Hence an early loss of galvanic as well as faradic

reaction is an important sign of neuritis. As the nerve and muscle recuperate, the galvanic irritability increases. After a time, if the paralysis is great, contractures occur. The feet are extended, the legs are flexed on the thighs, and are almost immovable, and the patient's condition is most pitiable.

Sensory Symptoms.—Numbness, hyperæsthesia, severe pains (dull and sharp), burning sensations, great tenderness, all occur, and are very marked symptoms. They are felt mostly in the feet, legs, and hands. Hyperæsthesia is usually followed by anæsthesia to touch and somewhat to pain and temperature. The transmission of these latter two sensations is delayed. The anæsthesia sometimes occcurs in patches, at other times diffusely over foot, leg, and hand. Muscular and articular anæsthesia are common, and in the sensory form are the dominant symptom, causing an ataxia of gait and station. The other special senses are not affected except in rare cases in which there is optic neuritis.

Vasomotor and Trophic Symptoms.—There is often œdema, sometimes redness of the skin; occasionally the epidermis of the soles and palms peels off. Glossy skin and profuse perspiration are rare. Eruptions and ulcers do not occur.

Mental Symptoms.—The most common mental disturbance is that so often seen in acute alcoholism, viz., a muttering delirium. This is associated with great general vital depression. If a true insanity develops, it also resembles, as a rule, alcoholic insanity or acute confusional insanity. The characteristic symptoms are a curious degree of forgetfulness, together with many and varying delusions rapidly succeeding each other. These often relate to the pains and paræsthesia from which the subjects suffer. They think that there are gloves on their hands or that something is on their feet. They often think that they have been out walking or riding. They are talkative, incoherent, and sleepless.

Organic Centres.—The bladder is occasionally affected for a short time, the other centres not at all. This freedom from involvement of the sphincters is an important characteristic of the disease in distinguishing it from myelitis.

From the foregoing it will be seen that the dominant symptoms are paræsthesia, pains (burning, lancinating, and dull), muscular tenderness, some anæsthesia, paralysis affecting especially the lower extremities and causing *foot drop*, muscular wasting, with degeneration reactions; with no involvement of the sphincters; sometimes peculiar mental disturbances.

The sensory or pseudo-tabetic type of multiple neuritis is caused less often by alcohol and more often relatively by diabetes

and the metallic and infectious poisons. Arsenic given medicinally in doses of one-sixth of a grain or more may cause such a neuritis. Multiple neuritis from lead is not often seen in painters, but usually when the poison is taken in larger doses, as in snuff takers. The general course of sensory neuritis is much like that of the paralytic form, but there is less paralysis, and on the other hand there are more of the burning, tearing pains, a greater degree of anæsthesia, with a very decided muscular anæsthesia causing symptoms of a subacute locomotor ataxia. The paresis, muscular wasting, trophic changes, such as shedding of the epidermis and electrical reactions, serve to distinguish the disease. A double facial paralysis sometimes complicates this type.

Endemic and Epidemic Types (*Beriberi or Kakke, Ignipedites, Acrodynia, Malarial Multiple Neuritis*).—Beriberi or endemic multiple neuritis is seen in this country rarely, and only by accident. Beriberi is the Indian name; kakke, meaning "the leg disease," is its Japanese name. Ignipedites is a name given by Indian physicians to probably the same disease. French physicians gave the name of "acrodynia" to an epidemic disease which prevailed in France and the Crimea in the early part of this century. It was probably multiple neuritis. There are various types of this disease, in some of which the neuritic symptoms seem subordinate to those of other organs. The forms described by Scheube and Taylor are:

The acute pernicious, the acute or subacute benign, the atrophic or dry, and the dropsical or wet.

The symptoms generally resemble those of multiple neuritis, as already described, plus œdema, extensive serous effusions, and gastro-intestinal disorders. The paralysis affects especially the lower extremities, but in beriberi there seems to be an especial tendency also to involvement of vasomotor and visceral nerves.*

Acute Pernicious Multiple Neuritis.—There is a form of multiple neuritis which comes on suddenly, progresses rapidly, and

* Malarial Multiple Neuritis.—Jamaica seems to be the only place in which the malarial poison produces an endemic neuritic paralysis (Strachan), and the causation in these cases is not yet demonstrated. Dr Strachan's description of the symptoms of what he terms malarial peripheral neuritis shows it to be quite extensive, often involving trunk and cranial nerves, and accompanied by much pain and wasting. Cramps and skin eruptions are often noted, complications that do not occur in the ordinary types. There are sporadic forms of multiple neuritis occasionally seen in this country, but it is yet to be proved that the malarial plasmodium can alone cause neuritis. It is more likely that it acts only in conjunction with some other toxic condition.

causes death in a few days or weeks. These cases usually show the ordinary symptoms of neuritic paralysis, with final involvement of the cardiac and respiratory nerves, causing death. The agent in these cases is apparently of the nature of sepsis. The neuritis is interstitial and hemorrhagic. Other cases of acute pernicious multiple neuritis take the form of acute ascending or Landry's paralysis. Here there are few sensory symptoms, no electrical changes or atrophy. The disease is due to an infectious poison which over-

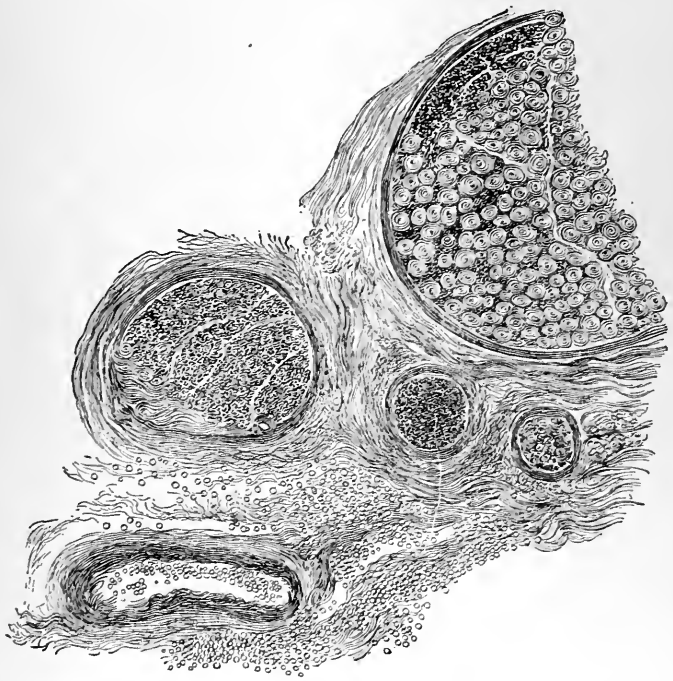


Fig. 51.—Diphtheritic Neuritis, Chiefly Interstitial (Siemerling); with secondary degeneration.

whelms the system before it has time to set up any inflammation or organic change. In these cases the anterior-horn cells of the spinal cord are also involved and the disease is one that attacks the whole peripheral motor neuron.

Pathology.—In multiple neuritis the disease affects the periphery of the nerves most, and extends up, very rarely reaching the roots. The anterior tibial and musculo-spiral nerves on the two sides are oftenest and most diseased. The process when mild in grade resembles a secondary degeneration following section of the nerve. In severer cases there is evidence of interstitial inflammation as well

as degeneration (Fig. 51). This process, however, varies in degree at different points of the nerve's course. Hence it has been called segmental or disseminated neuritis. In some of these cases and in all acute pernicious cases there is still more interstitial inflammatory change; small hemorrhages occur, exudation takes place, and collections of leucocytes about the vessel walls and among the nerve fibres are seen (Fig. 52). The muscles supplied by the diseased nerves undergo atrophy. This is usually simple and non-inflammatory. But sometimes there is an interstitial myositis with exudation compressing the fibres (Senator).

If the disease progresses, the nerve fibres degenerate and their

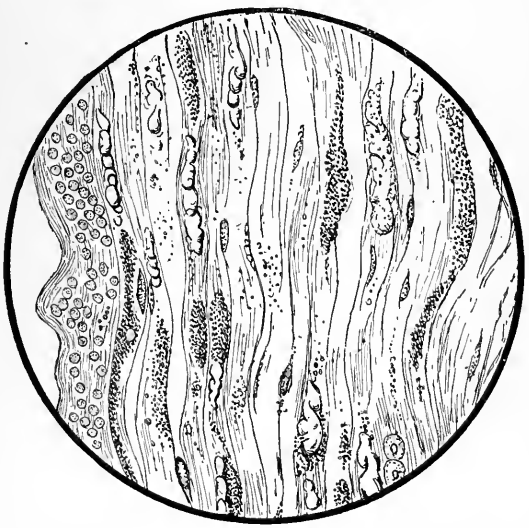


FIG. 52.—LONGITUDINAL SECTION OF A NERVE IN MULTIPLE NEURITIS, showing rich proliferation of nuclei (Leyden). The process here is inflammatory as well as degenerative.

place is taken by connective tissue, and the same process occurs in the muscles.

The spinal cord when examined by the help of Nissl and Marchi stains shows some involvement. The anterior-horn cells undergo the same degeneration as that which occurs when the nerve is cut across, and slight areas of degeneration are found in the posterior and lateral columns. The changes are very slight compared with those in the nerves and in the writer's opinion are secondary.

It will be seen, therefore, that in multiple neuritis there may be: (1) Simple degeneration; (2) degeneration with some evidences of interstitial neuritis (degenerative neuritis); (3) decided interstitial neuritis with degeneration of nerve fibres.

Diagnosis.—Multiple neuritis must be diagnosticated from diffuse or transverse myelitis, anterior poliomyelitis, locomotor ataxia, spinal meningitis, and hemorrhage and Landry's paralysis. Practically, diffuse myelitis is the disorder from which it has oftenest to be distinguished. From this it is recognized, first, by investigating the cause and onset. Neuritis begins more slowly and with sensory prodromata; it affects the legs and feet, especially the extensors, and if it ascends it skips the hips and trunk and attacks the forearms. There is more muscular atrophy than in myelitis; the knee jerks are absent. It progresses more slowly, and after four or eight weeks gradually regresses. Electrical degeneration reactions are more varied and decided. There are tenderness over the muscles and nerves and peculiar burning, darting pains. The cutaneous anæsthesia, if present, is not so extensive and complete, as a rule, while muscular anæsthesia is more decidedly marked. There is very rarely involvement of the sphincters or bedsores. There may be belt-like constrictions felt round the extremities, but not around the waist. The gradual improvement of the paralysis and atrophy and eventual recovery confirm the diagnosis of neuritis. The presence of neuritis of the cranial nerves would strengthen the theory of a general neuritis.

From poliomyelitis it is distinguished by the presence of pain and other sensory symptoms, the early fall in galvanic irritability, the age of the patient, and the etiology.

From locomotor ataxia, neuritis is distinguished by its more rapid onset, the presence of paralysis and atrophy of muscles, paresis, with degeneration reactions, and the absence of involvement of the organic centres and pupils.

Spinal hemorrhage usually leads soon to a secondary diffuse myelitis easily distinguishable from neuritis by the characters above given. Here there is also usually pain in the back. Spinal meningitis is associated with characteristic pain, tenderness, and stiffness along the back. Acute ascending paralysis in its typical form shows but very slight sensory disorders, and no wasting or change in electrical irritability.

The complication of multiple neuritis and myelitis or posterior sclerosis is possible, but is very rare. In the former case the ordinary symptoms of myelitis are added to those of neuritis. In locomotor ataxia there is often some nerve degeneration and occasionally neuritis. The nerve degeneration probably causes only slow atrophic changes and paresis; the neuritis causes pains, anæsthesia, skin eruptions, and local trophic disorders.

Prognosis.—Alcoholic multiple neuritis is a serious disease, be-

cause of its associated conditions. Nearly one-half of my patients have died, mainly because they continued the use of alcohol after paralysis appeared. They do not die of neuritis, but of alcoholism or of phthisis. Other forms of neuritis rarely cause death. The great majority recover almost completely. It may be from six months to two years before all symptoms disappear. The average time is about a year.

Treatment.—The patient needs, first of all, rest in bed. The limbs are often extremely tender and the patient's pains excruciating. To relieve these the legs may be painted with menthol and enveloped in cotton batting. In other cases flannels wrung out in hot water and renewed every two hours give relief. Internally phenacetin, antipyrin, or other coal-tar products may be given for the pains. Fluid extract of ergot in doses of ʒ i. to ʒ ii. repeated in three hours sometimes relieves pain. In the early stages, salicylate of soda in doses of gr. xx. every two or three hours is recommended. If there is a great deal of depression from alcoholic poisoning, strychnine, gr. $\frac{1}{60}$ q. 3 h., and aromatic spirits of ammonia, ʒ ss. q. 3 h., should be used.

There is no drug which really cuts short the process. The best measures for this purpose are rest, thorough cleansing of the alimentary tract, abstinence from alcohol, and a nourishing diet.

After the acute stage is passed the labile galvanic current occasionally interrupted may be applied, 5 to 10 ma. for ten minutes three times weekly. Later, by the sixth week, the faradic current, massage, and careful exercise should be given. At this time or earlier (third week), strychnine, iodide of potassium, arsenic in small doses, and tonics may be given. In old cases in which a great deal of paralysis and contracture have occurred, forcible extension of the limbs, the use of splints and rubber muscles, are needed. With patience and perseverance the worst cases can eventually be brought to a complete recovery.

Complicating Forms of Neuritis and Neuritic Degeneration.—Neuritis and neuritic degeneration complicate many diseases, but they especially mark and modify subacute and chronic rheumatism, locomotor ataxia, diabetes, paralysis agitans, wasting diseases, and old age.

A *neuritic* degeneration almost always affects the nerves in the neighborhood of an old rheumatic joint. The chief result of this is to produce wasting and some paresis of the muscles moving the joint (Pitres and Vaillard). The process is a reflex atrophy (see Arthritic Muscular Atrophy).

In locomotor ataxia, parenchymatous nerve degeneration is very

often present. It does not produce the cardinal symptoms of this disease. It does, however, cause some of the anæsthesia, paræsthesia, muscular atrophy, skin dystrophies, and visceral crises.

In diabetes, the neuritis takes the form of the sensory type of multiple neuritis, and causes symptoms like those of locomotor ataxia. The patient has sciatic pains, burning or numb feet, loss of tendon reflex, ataxia. The upper extremities are rarely affected.

In Wasting Diseases and Old Age.—In various wasting diseases, such as phthisis, cancerous cachexia, long-continued fevers, marasmus, and in senility, a simple parenchymatous degeneration of nerves, with atrophy, occurs (Arthaud, Köster, Jappa). The symptoms caused by these changes are very slight. They contribute to the weakness and wasting. In old age, the atrophy of the nerves is one cause of the lessened sensibility and activity of the skin and its underlying muscles.

TUMORS OF NERVE.

These consist of:

1. Nerve hyperplasia.
2. True neuromata. { Single. { Benign. / Malignant.
3. False neuromata. { Multiple. { Benign.

1. *Hyperplasia* or hypertrophy of nerve trunks is very rare. Generally the increase in size is, in fact, due to increase of the interstitial connective tissue. Sometimes there is an increase in the number of fibres and thickening of the myelin sheath.

2. *True neuromata* are also very rare, and occur almost exclusively on spinal nerves. In some there is an increase in medullary fibres; in others only an increase of non-medullated fibres, *i.e.*, only the axis cylinders and neurilemma increase. They occur either singly or multiply. *Multiple neuromata* are generally of a neuro-fibromatous character.

Neuro-fibromata when multiple may affect the subcutaneous nerves and form growths known as *fibroma molluscum*.

Nerve fibres united in a mass by hyperplastic connective tissue form what are called *plexiform neuro-fibromata*. Multiple neuromata may be true neuromatous growths.

True neuromata are usually small, ranging from 1 cm. (two fifths of an inch) to 6 cm. in diameter. They may be much smaller or larger. Neuromata are usually few in number, or at least there is only a local multiplicity of tumors. Multiple (true) neuromata may, however, be very numerous. Gowers estimates in one case that as

many as one thousand were present. Even larger numbers have been observed.

3. *False Neuromata.*—This term is applied to the various nerve tumors in which a fibroma, myxoma, glioma, sarcoma, carcinoma, or syphiloma grows upon or in the nerve. Fibro-neuroma is the most common form; glio-neuroma has been observed on the auditory nerve. Syphiloma occurs only on the intracranial or intraspinal nerves. Carcinoma of nerves may be primary, but is generally secondary, and is of scirrhous or medullary type, rarely the colloid. Leprous neuritis sometimes forms neuro-fibromatous swellings.

Tubercula dolorosa are simply small false neuromata situated subcutaneously on the ends of the sensory nerves. They vary much in histological structure.

Malignant Neuromata.—A few cases, about thirty in all, have beeen observed of multiple malignant neuromata. Trauma and hereditary influence are the etiological factors. The great nerve trunks are oftenest affected, the median, sciatic, and crural ranking first. The tumors start from the perineurium; they are at first spindle shaped, and may grow very large. Sarcomatous cells are oftenest found in them; but they may be myxomatous, fibromatous, or mixed.

Etiology.—Three general causes exist for the production of neuromata, viz.: 1. A hereditary or a neuropathic predisposition; this tends to cause the true, the multiple, and the plexiform neuromata. 2. Injuries, surgical operations; these cause especially the fibro-neuromata of which the *amputation neuroma* is an example. 3. Diathetic, *e.g.*, tuberculous, influences and whatever produces the various tumor formations, sarcoma, carcinoma, form the third etiological factor. Neuromata of the plexiform type are often congenital. Multiple neuromata may develop early in life. Men are far more subject to multiple neuromata than women.

Symptoms.—Neuromata often cause no symptoms. Perhaps the most frequent evidence of their presence, however, is pain and some tenderness. The pain is exacerbating, and may be stopped sometimes by pressure on the nerve above the tumor. Paræsthesia, anæsthesia, paralysis, and reflex spasm may be present. Some forms of intractable headache are possibly due to multiple neuromata. Multiple and plexiform neuromata cause symptoms less often than a single larger neuroma. A neuroma on the pneumogastric or other splanchnic nerve may cause severe symptoms. Multiple (true) neuromata may last for years and cause no serious inconvenience. Malignant neuromata cause always such symptoms as would naturally follow irritation and compression of a nerve.

The *diagnosis* of neuroma can be certainly made only when the tumor can be felt. In other cases, by exclusion a fairly certain conclusion may be reached.

True neuromata are often multiple; the false rarely. Idiocy, heredity, neuropathic constitution, scrofula, would all favor the view of the disease being a true neuroma.

The *treatment* is essentially surgical. Internal medication and external applications are of little value. Strong galvanic currents, mercury, and the iodides in large doses may be tried in true neuromata and fibro-neuromata. Both true and false neuromata may return after extirpation.

CHAPTER VIII.

MOTOR DISORDERS OF SPECIAL NERVES.

The general distribution of the paralyses from injury or disease of the different motor nerves is shown in the accompanying table of cases. I am indebted to my friend, Dr. Wm. P. Wilkin, for the larger part of the work of compilation:

Cranial-nerve paralyses (including 19 occurring in locomotor ataxia)	92
Spinal-nerve paralyses	113
Multiple neuritis	68
	273
Of the cranial nerves:	
Ocular (third, fourth, sixth)	28
Fifth	2
Seventh	46
Tenth and eleventh	4
Of the spinal nerves:	
Arm	97
Thigh and leg	16

It will be seen that the brachial plexus and its branches are oftenest affected, next the seventh cranial nerve, then the ocular nerves, and last the lumbar and sacral plexus. This of course represents the frequency in a nurologist's experience. In general practice and especially in surgical practice there would be fewer cases of cranial-nerve disease and more of lumbar and sacral palsies.

The Ocular Muscles.

Anatomy.—It will help the student in learning the diseases of the cranial nerves if their points of origin and general relation are shown, as in the accompanying illustration (Fig. 53).

The motor nerves of the eye are:

(*a*) The third or oculo-motorius, supplying the internal, superior, and inferior recti, inferior obliquus, the levator palpebræ, the ciliary muscle, and constrictor of the iris.

(*b*) The fourth or trochlearis, suppyling the superior oblique.

(*c*) The sixth or abducens, supplying the external rectus.

(*d*) The sympathetic, consisting of fibres from the upper cervical

nerves to the dilators of the iris, to its blood-vessels, and to Müller's muscle.

Motor fibres from the nucleus belonging to the third nerve run out with the fibres of the seventh (Mendel), supplying the orbicularis palpebrarum.

The third and fourth nerves arise from a series of nuclei in the floor of the aqueduct of Sylvius. They leave the brain at the an-

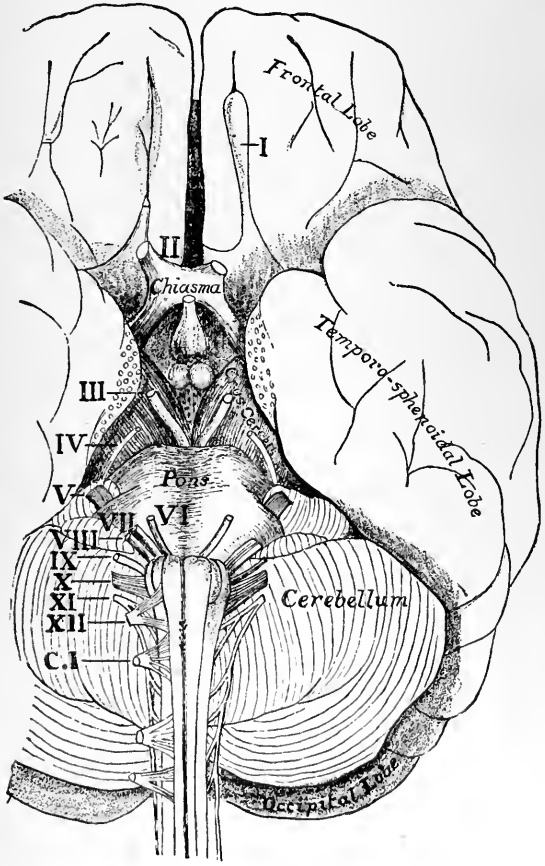


FIG. 53.—SHOWING THE APPARENT ORIGIN OF THE CRANIAL NERVES.

terior edge of the pons. They run in the cavernous sinus and enter the orbit through the sphenoidal fissure.

The sixth nerve arises from a nucleus in the floor of the fourth ventricle. It emerges at the posterior edge of the pons, runs in the cavernous sinus, and enters the orbit through the anterior lacerated foramen.

The nuclear gray matter from which these nerves arise is made

up of a series of nests of cells and each pair supplies a different set of muscles of the eye, as shown in the diagrams (Figs. **56** and **57**).

The nucleus of the sixth lies farther back in the floor of the medulla, but it belongs to the same serial deposit of gray matter and represents the continuation of the anterior horn of the spinal cord (Fig. 56).

The motor nerves of the eye, third, fourth, and sixth, are closely connected with each other and other nerves by a long commissure, the *posterior longitudinal bundle*.

The fibres of the third and fourth nerves pass to their nuclei on the same side, then decussate and pass up in the inner part of the

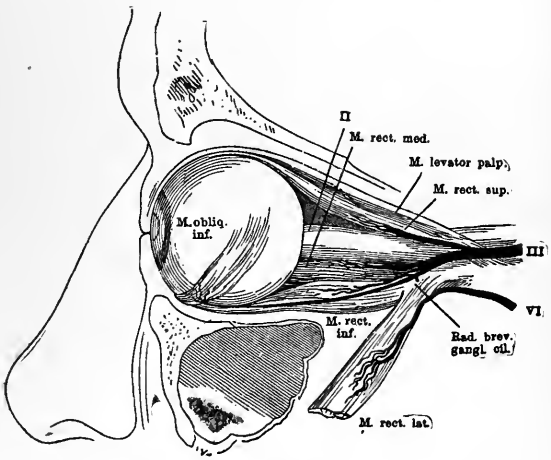


FIG. 54.—SHOWING DISTRIBUTION OF THIRD AND SIXTH CRANIAL NERVES.

crusta to the frontal part of the central convolutions of the cortex. A few fibres decussate and enter the nuclei of the opposite side. They are connected with the internal rectus nucleus.

The fibres of the fourth nerve almost entirely decussate, running forward a long distance before they finally reach their nuclei. Thus it appears that the fourth is the only cranial nerve except the optic which largely decussates before reaching its nucleus. However, those fibres of the third which supply the internal rectus also decussate, as already stated.

The arrangement of the nuclei is believed to be as follows, the upper on the list being anterior:

Median line.

III. N.	Sphincter iridis, Ciliarius. Levator palp., Rect. int. Rectus superior, Rect. inf. Obliquus inferior.
IV. N.	Obliquus superior.
VI. N.	External rectus.

In order to understand the peculiarities of eye palsies, to be described later, the relations of the sixth to that nucleus of the third nerve which innervates the internal rectus must be understood. In turning the eyes to one side, these two nuclei and their nerves act together, causing the external rectus of one eye and the internal rectus of the other to contract at the same time. The impulse from the brain which does this decussates and acts first upon the sixth, and through this upon the external-rectus nucleus of the same side. The impulse from this nucleus then goes to the third-nerve fibres of

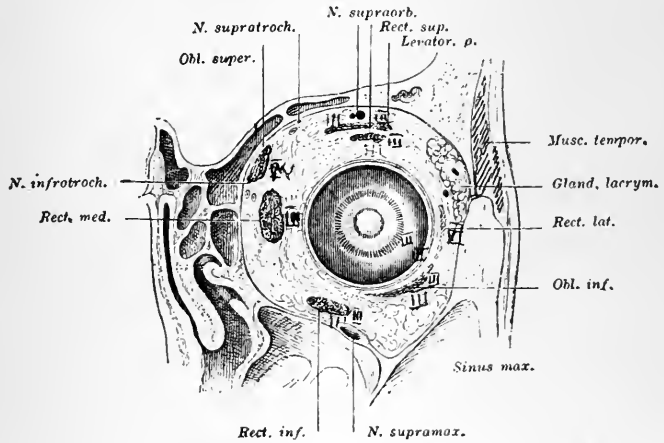


Fig. 55.—The Attachment of the Eye Muscles to the Globe (Merkel).

the same side and thence to the internal rectus. This can be better understood by the diagram, Fig. 57.

Thus lesions in the brain at (*a*) cause paralysis of the sixth nerve of the opposite side and internal rectus of the same side. The eyes turn toward the side of the lesion.

Lesions in the pons at (*b*) cause paralysis of the sixth on the same side and internal-rectus nucleus of the opposite side The eyes turn away from the side of the lesion.

The eye muscles move the eyeball in the following way:
The superior rectus elevates the eyeball.
The inferior oblique rotates out and up.
The inferior rectus depresses the eyeball.
The superior oblique rotates out and down.
The superior and inferior oblique, acting together, rotate inward.
The external rectus rotates outward.
The internal rectus rotates inward.
The rectus internus, rectus superior, obliquus inferior, rotate upward and inward.

The rectus internus, rectus inferior, obliquus superior, rotate downward and inward.

The rectus externus, rectus superior, obliquus inferior, rotate out and up.

The rectus externus, rectus inferior, obliquus superior, rotate out and down.

The movements of the eyeball are made by the simultaneous action of several muscles. Most of them act as their names indicate. But the oblique muscles help to depress and elevate, and then help to rotate in or out according as the internal or external rectus is acting.

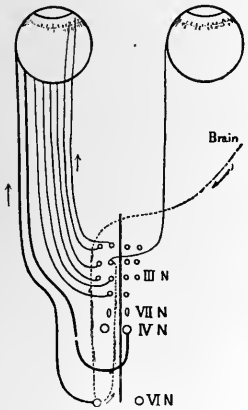


FIG. 56.

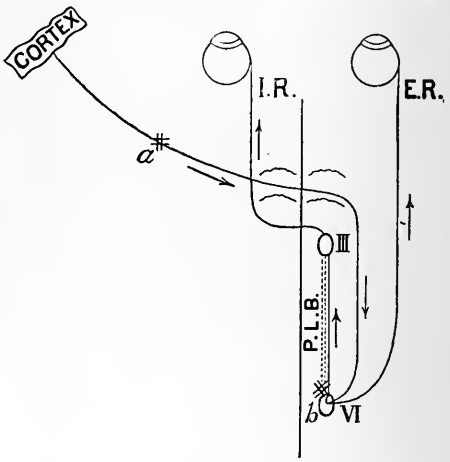


FIG. 57.

FIG. 56.—DIAGRAM SHOWING THE ARRANGEMENT OF THE NUCLEI OF THE MOTOR NERVES OF THE EYE, AND THE DECUSSATIONS OF THE FOURTH AND INTERNAL RECTUS BRANCH OF THE THIRD NERVE.

FIG. 57.—DIAGRAM SHOWING THE PROBABLE RELATIONS OF THE NUCLEI OF THE SIXTH AND OF THE INTERNAL RECTUS BRANCH OF THE THIRD TO THE BRAIN. *P.L.B.*, Posterior longitudinal bundle.

The cortical centres for the eye muscles are not positively known. Lesions in the inferior parietal lobule sometimes cause paralysis of the third nerve. Lesions of the posterior part of the prefrontal lobes also sometimes cause eye palsies, especially conjugate deviation, and the dominant are in the prefrontal lobe at the base of the upper and middle frontal convolutions.

As the optic nerve is the special sensory nerve of the eye, so the third, fourth, sixth, and part of the seventh nerves are the motor nerves. By means of the optic nerve and its receptive and refractive apparatus, the form, color, movement, and, to some extent, relations and distance of objects are determined. The motor nerves adjust the eye to near and distant objects, inform us as to size and distance, and enable us to follow moving objects and to shift the gaze readily. They also assist in protecting the eye against injury.

General Symptoms.—It is impossible always to disassociate diseases of the oculo-motor nerves from those involving their nuclei. Hence we must study here really the affections of the whole neurons. These are:

1. Paralyses or ophthalmoplegias, which may be acute, chronic, or progressive. 2. Pareses or amyosthenic states, called ordinarily muscular asthenopias. 3. Spasms, such as strabismus, nystagmus, and blepharospasm.

There are many special terms which are used to indicate the peculiar effects of various paralyses and spasms of the ocular muscles and nerves, and some of these I will define here:

Erroneous projection is a condition in which the patient is unable to judge exactly of the relation of external objects to the body; for this relation is determined by the movements of the ocular muscles, and, these being weak, wrong sensations are conveyed to the brain. Vertigo may result from this disturbance of muscular sensation.

Diplopia or *double vision* is a condition due to the erroneous sensation resulting from eye-muscle palsy, and to the fact that the images of the object fall upon non-corresponding retinal fields. Diplopia is simple or homonymous when the false image is seen on the same side as the affected eye. When a red glass is placed over this eye two images are seen, the red one being on the side of the eye involved. Diplopia is heteronymous or crossed when the false image is on the side opposite to the sound eye.

Conjugate deviation of the eyes is a condition in which both eyes turn strongly to one or the other side. It may be paralytic or spasmodic. The mechanism is a complicated one and not perfectly understood. In general, destructive lesions of the brain cause a paralytic deviation toward the side of the lesion, and irritative or compressing lesions the opposite effect. Destructive lesions in the pons cause a deviation away from the side of the lesions. The palsy then involves the sixth cranial nerve and the branch to the internal rectus from the third. The sixth-nerve nucleus is the dominant one, and impulses from the brain go to it first (see Fig. 57).

In diseases of the motor nerves of the eye it is found that the paralyses occur in various ways, which may be best grouped as follows:

Ophthalmoplegias.

1. Paralyses of the third nerve. } Acute.
2. Paralyses of the fourth and sixth nerves. } Chronic.
3. Progressive paralysis of all or part of these nerves.

The Ophthalmoplegias.

I. Paralysis of the Oculo-Motorius or Third Nerve—*Etiology.*—The commonest causes are exposure to cold, and **syphilis.** Other causes are basal meningitis, intracranial tumors, injuries, compression from orbital tumors, the diphtheritic poison, and excessive exposure to light; excessive use of tobacco, and alcohol, morphine, or other poisons may be a cause. Temporary palsy sometimes occurs in migraine, or it may take the place of an attack of migraine. A palsy of some of the muscles supplied by the third is sometimes caused by cerebral lesions involving the inferior parietal lobule. Partial palsies also occur in locomotor ataxia and in certain primary muscular atrophies. The common causes, however, are, as stated, rheumatic influences and syphilis.

Fig. 58.—Double Ptosis.

There occurs, in rare cases, an acute inflammatory degeneration of the nuclei of the ocular-muscle nerves similar to acute anterior poliomyelitis. This condition has been called "polio-encephalitis superior" or upper bulbar palsy.

Symptoms.—When all the muscles supplied by the third nerve are paralyzed, there is dropping of the lid (ptosis, Fig. 58); the eye can be moved only outward and downward and inward; there is therefore divergent strabismus and double vision (diplopia). The pupil is somewhat dilated and does not contract to light, owing to paralysis of the constrictors of the iris; and there is loss of power of accommodation, so that the patient cannot read print close to him.

The patient suffers much annoyance from the lid drop and the double vision, and there are sometimes vertigo and photophobia. Only one nerve is involved at a time as a rule. The various eye muscles supplied by the third are rarely all attacked. The levator may escape almost entirely; the ciliary muscle and iris may also be but slightly involved; but these latter muscles are never involved alone in ordinary types of the disease.

The affection usually runs a subacute course, lasting but a few weeks. Functional palsies last but a few days; syphilitic palsies are usually temporary (one to three weeks), but may relapse or become extremely obstinate. Periodical palsies occur every year or six months or even oftener; they last a few days or weeks and are accompanied at first by some pain. They continue to recur for years. They may be associated with attacks of migraine.

In diphtheritic eye palsies the first three or four nuclei of the series making up the origin of the third nerve are oftenest affected, causing paralysis of accommodation, paralysis of the iris and of the internal rectus, the three muscles concerned in accommodating the eye to near objects.

Pathology.—In the rheumatic palsies there is a low grade of peripheral neuritis, and the same is true of most diphtheritic and other palsies of infectious origin. In syphilitic and tabetic palsies

FIG. 59.—SHUTTER FOR TESTING PUPIL REFLEX. The apparatus admits of using colored glasses for testing hysteria and malingering; also of the use of a Maddox prism for testing the eye muscles.

there is usually a specific basilar meningitis involving the nerve roots. The meningitis may be slight or may amount to gummatous deposit. In functional and some periodical palsies there is a vaso-motor disturbance causing congestion or anæmia or perhaps simply inhibition of the nuclear centres. Some periodical palsies have been found to be due to small tumors involving the nerve root. In rare cases there is primary muscular atrophy of the eyeball nerves, or primary degeneration of the nuclear centres, or a cerebral lesion. The nuclear inflammation forming "polio-encephalitis superior" is a disease probably infectious and quite similar to anterior polio-myelitis.

Diagnosis.—One must first determine how extensively the muscles supplied by the third are involved.

If only the levator palpebræ, there is simply falling of the lid. If the eyeball muscles are involved, we get the following symptoms. They are:

Limitation of movement of the globe.

Strabismus and secondary deviation.

Erroneous projection.

Double vision or diplopia, which is either simple or crossed.

Paralysis of the iris or iridoplegia and of the ciliary muscle or cycloplegia.

Concentric limitation of the visual field.

All these points must be tested, but the detailed knowledge of them is best gained by consulting ophthalmological works.

The extent of involvement of eye muscles can generally be sufficiently tested by making the patient move the affected eye in various directions, and by testing for accommodation and for the pupillary reaction to light.

Paralysis of the iris, or iridoplegia, and of the ciliary muscle—cycloplegia.

The motor fibres of the third nerve to the iris supply the sphincter, and when paralyzed there are dilatation and immobility of the pupil, a condition known as *mydriasis*. Fibres from the same nucleus innervate the ciliary muscle, and the iris and this muscle are usually paralyzed together. Paralysis of the ciliary muscle is called *cycloplegia*. In this latter condition there is loss of power of accommodation. Iridoplegia and cycloplegia are usually due to local disease of the eye or to the use of mydriatic drugs. Occasionally they are observed after diphtheria or in multiple sclerosis. It may occur in locomotor ataxia, and in disease of the lower cervical cord involving the cilio-spinal centre, which when destroyed causes a myosis.

Paralysis of the levator palpebræ, causing *ptosis*, is sometimes seen alone, but usually other branches of the third nerve are involved. A functional palsy of the lids sometimes occurs in anæmic and nervous people at the time of waking. It is a temporary *morning* or *waking ptosis*.

Paralysis of the sympathetic fibres of the eye causes contraction of the pupil (myosis) from the unopposed action of the third nerve. There is also a slight prominence of the eye and slight ptosis from an involvement of the nerves that supply Müller's muscle. The pupil does not dilate when the skin of the cheek or neck is irritated. This is a condition known as loss of skin reflex. In locomotor ataxia there is often a rigidity of the constricting fibres

of the iris, while the ciliary muscle continues to act. The pupil is then small and does not respond to light, while it does respond to accommodation. This is known as the *Argyll-Robertson pupil.*

II. Paralysis of the Fourth Nerve.—This is a rare affection and not always easily detected. The causes are much the same as those of palsy of the third nerve.

The symptoms are slight convergent strabismus when the eye is moved downward and diplopia on looking down. There is defect in the movements of the eye downward and outward.

Paralysis of the sixth nerve (abducens) is the most frequent of eye palsies, and occurs especially often in syphilis and in locomotor ataxia. It causes convergent strabismus and double vision.

III. Progressive Ophthalmoplegia (*Progressive Upper Bulbar Palsy*).—Besides the palsies already described, there occur certain forms which have a peculiar origin and course. They begin slowly, as a rule, and steadily progress. In some cases only do they reach a certain stage and then remain chronic. The term "progressive" applies fairly well to them. They often affect the third, fourth, and sixth nerves together. In accordance with the muscles invaded, these palsies are called *external, internal, partial,* and *total.* Thus if those branches of the third nerve supplying the iris and ciliary muscle are involved alone, it is ophthalmoplegia interna; if the other branches are involved, it is called ophthalmoplegia externa.

Definition.—Progressive ophthalmoplegia is a degenerative disease of the nuclei of the motor nerves of the eye. It is in most cases the same disorder as of bulbar paralysis and progressive muscular atrophy.

Etiology.—It develops between the ages of fifteen and forty, but may occur later. The sexes are equally affected. Lead, diphtheria, traumatism, syphilis, appear sometimes to be the cause. It may complicate locomotor ataxia; more often it forms part of progressive muscular atrophy.

The *symptoms* are often not noticed until the disease is far advanced. The vision is not disordered, and there is only a gradual limitation of mobility of the eyeball. A slight drooping of the lids, causing a sleepy look, or a slight squint, usually divergent, is noticed. Then upon examination it is found that the eye cannot follow the finger, except to a slight extent. This peculiar physiognomy is known as the "Hutchinson face" (see Fig. 60). The iris reacts to accommodation and light usually. Double vision may be present. Usually the patient accustoms himself to monocular vision. The disease lasts a long time, and it may become stationary. If complicated with progressive muscular atrophy, however,

the course is relatively rapid, death occurring from the latter disease in two or three years.

Pathological Anatomy.—In all progressive cases there is a degenerative atrophy of the nuclear cells. In a few rare cases no lesion has been found, and in a few stationary cases the anatomical change is that of neuritis.

The *treatment* is that for the disease which it complicates or the condition which causes it. That is to say, it is the treatment for locomotor ataxia, progressive muscular atrophy, syphilis, or lead

FIG. 60.—SHOWING "HUTCHINSON FACE."

poisoning. Iodide of potassium, strychnine, arsenic, nitrate of silver, and phosphorus may be given. Electricity is of very doubtful value, and only the galvanic current would be indicated. General tonic measures and rest to the eyes should be employed.

MUSCULAR ASTHENOPIA AND MUSCULAR INSUFFICIENCIES.*—This is a term employed to indicate a lack of equilibrium of the muscles of the eye, as a result of which the visual axes cannot be kept parallel without an effort. This effort is often unconscious, and shows itself only by a ready tiring of the eye on attempting to read, or by the production of headaches and cerebral paræsthesiæ. Examination of the eye by means of prisms reveals the special character of the trouble.

When the eye muscles act normally the condition is called one

* Partial ophthalmoplegia occurs also in myasthenic paralysis.

of *orthophoria*. When some of the muscles are weak it is called *heterophoria*. There are various forms of heterophoria, viz.: esophoria, a tending of the visual lines inward, from weakness of the externi; exophoria, a tending of visual lines outward; hyperphoria, a tending of the visual line of one eye above its fellow.

The condition is tested in various ways. The simplest is this: Refractive errors having been corrected, a series of prisms is placed over the eye, at first with the base inward, while the patient looks at a candle twenty feet distant. The prisms are increased in strength until the patient can no longer coalesce the images. The degree of prism is noted, and this indicates the strength of abduction or of the externi. The same process is gone through with for the interni, the base of the prism being out. The externi should overcome a prism of about 8°, the interni one of 23° to 25° or more. There are great individual variations, and there is also considerable variation in individuals.

The above tests measure the amount of abduction and adduction.

To test the presence of heterophoria, the writer uses the Maddox double prism held in a frame. The line where the bases of the two prisms unite is brought directly over one eye, and is held there in a perfectly horizontal position while the patient looks at a candle twenty feet away. A red glass is at the same time held over the other eye. With the eye covered by the double prism the patient sees double, one flame being above the other; with the other eye he sees a red flame lying just between the two white ones. If the red flame is directly in a vertical line, there is orthophoria; but if it lies to one side or the other there is exophoria or esophoria according as the red flame was on the side opposite to the eye covered with red glass or on the same side. If heterophoria is found, prisms are placed over the eye until the three lights are in a vertical line. The number of the prism required to correct the heterophoria indicates its extent in degrees. By changing the double prism so that its common base line is vertical, the test for hyperphoria can be made.*

Muscular asthenopia is said to cause a disturbance of vision, vertigo, migraine, cerebral paræsthesia, and pains in the head, more particularly in the occipital and cervical region. It is believed to be a possible factor in producing choreic twitchings in the face. In neurasthenic persons it may cause a wider range of nervous symptoms. It is said to be an essential factor in causing epilepsy, chorea, and hysteria. The author cannot accept this latter view, and believes that the importance of muscular asthenopia in causing

* More elaborate and exact methods have been devised by Dr. Stevens and are employed by ophthalmologists.

general nervous symptoms is not great. Much of it, if not all, may be relieved by correcting refractive errors and by helping the general health of the patient.

The treatment of it, after all myopia, or hypermetropia, or astigmatism, if present, is relieved, consists in building up the general health, the systematic use of prisms for training the muscles, the wearing of proper glasses. Some advise graduated or complete tenotomies according to the method of Stevens.

Spasmodic Diseases of the Ocular Muscles.

These are: (1) Conjugate deviation from spasm; (2) irregular and associated spasms from convulsive and irritative brain disorder; (3) nystagmus.

Spasmodic conjugate deviation occurs from an irritating lesion of the ocular nuclei or of the brain in its cortical motor areas and tracts. Irregular spasmodic movements occur in meningitis, hydrocephalus, and in lesions involving the semicircular canals. Peculiar associated spasms occur in hysterical attacks. Various spasmodic movements and contractions of individual eye muscles occur from ocular disease, errors of refraction, muscular weakness, and paralysis of certain eye muscles.

Rhythmical spasm or *nystagmus* occurs as the result of hereditary visual weaknesses and refractive errors of various kinds, in albinoes, and in chronic hydrocephalus. It is found usually in neurotic cases associated with ocular defects, in multiple sclerosis, and sometimes in epilepsy, chorea, hysteria, neurasthenia, and insanity. It occurs in certain degenerative nervous disorders such as disseminated sclerosis, hereditary ataxia, tumors, especially of the cerebellum, and other focal lesions, and in meningitis. It occurs in miners, and is called miners' nystagmus. It may be a reflex symptom from a remote irritation.

In nystagmus the oscillation of the eyeballs is usually lateral. It may be brought out when slight in degree by causing the patient to look steadily to one side. Vertical and a kind of rotating nystagmus sometimes occur, and are due to much the same causes as those of lateral nystagmus.

Spasm of the levator palpebræ is sometimes seen and is usually tonic.

The above troubles are usually symptomatic, and their treatment depends upon correction of some local disease or cerebral neurosis.

The Motor Branch of the Fifth Cranial Nerve.

The anatomy of this nerve is described under the head of the neuralgias of the trigeminus.

The diseases of the motor branch of the trigeminus are rare, and generally symptomatic of some more general disorder.

Trismus (lockjaw) is the only important independent affection of this motor nerve. It is a tonic spasm of the muscles of mastication.

Etiology.—It occurs in infants, usually through infection from the umbilicus. It is then known as trismus nascentium. It forms part of the symptoms of tetanus and rabies. It may be symptomatic of brain disease, and forms one of the manifestations of the epileptic fit. There may be a reflex trismus from irritations of the teeth and jaw, and from gastro-intestinal trouble. Neuritis and hysteria are causes of trismus and it may be associated with a trigeminal neuralgia.

Symptoms.—There is, as shown above, a symptomatic, an infectious, a reflex, a neuritic, and a hysterical trismus. In all, the symptoms are very manifest. The jaws are firmly locked and the masseters and temporals stand out. If the disease is unilateral, which is rarely the case, the lower jaw is pushed over toward the sound side.

The *treatment* of both tonic and clonic spasms depends upon the cause. Symptomatically, morphine is to be given, and later the antispasmodics, such as the bromides and chloral. In rheumatic cases hot applications and diaphoretics are indicated.

The Facial Nerve.

Anatomy.—The facial nerve has its primary origin in a single nucleus deeply situated in the lower part of the pons (Fig. 61). It belongs to the same series of nuclei as the motor nuclei of the vagus, glosso-pharyngeal, and of the spinal accessory; in other words, it is a prolongation of the lateral horn of the spinal cord. It has not a nucleus common to it with the sixth, as is usually stated. Those fibres of the nerve which go to the orbicularis, however, appear to come from a nucleus in the third-nerve series and to reach the knee of the facial by the posterior longitudinal bundle (Mendel). The deep fibres of the facial take a tortuous course, passing inward, dorsally, then curving down and out around the nucleus of the sixth nerve (Fig. 62). The cortical origin of the seventh is in the lower part of the central convolutions, especially the precentral. The fibres pass down through the knee of the internal capsule and enter

the crusta at the inner side of the pyramidal or motor tract. They decussate and reach the nucleus. The nerve has its exit at the posterior edge of the pons, external to the sixth nerve. It then has to take a long course through a bony canal, during which it receives taste fibres from the second or third branch of the trigeminus (or the glosso-pharyngeal). These fibres leave the nerve at the chorda tympani, and join the lingual branch of the fifth nerve to supply taste to the anterior two-thirds of the tongue.

The facial nerve supplies motion to all the muscles of the face; to the stapedius, stylo-hyoid, buccinator, and platysma myoid. It

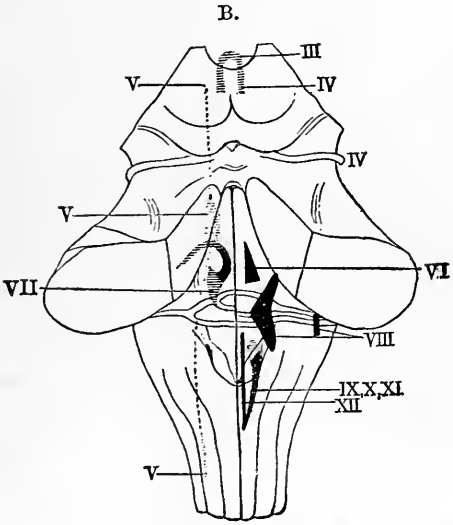


FIG. 61.—SHOWING THE POSITION OF THE CRANIAL NUCLEI IN THE MEDULLA.

also contains trophic and secretory fibres. It does not supply, however, the muscles of mastication.

The taste fibres of the facial nerve come in most cases from the second branch of the fifth via Meckel's ganglion, the large superior petrosal nerve, and geniculate ganglion. In other cases they come from the glosso-pharyngeal nerve via the ganglion petrosum, Jacobson's nerve, tympanic plexus, and geniculate ganglion (Fig. 63). Some think that the intermediary nerve of Wrisberg, which arises in the upper part of the glosso-phyaryngeal nucleus and connects with the geniculate ganglion, carries taste fibres to the facial.

The facial nerve being motor, its diseases are spasmodic and paralytic. The two common types are facial tic and facial palsy, but there are other minor forms.

The spasmodic disorders are (1) diffuse facial spasm or mimic tic and (2) spasm of single branches, including (*a*) blepharospasm and (*b*) nictitating spasm.

Facial Spasm (Mimic Tic).—This is a disease characterized by intermittent, involuntary twitchings of the facial muscles. It is always chronic and generally unilateral.

Etiology.—It is a disease of middle and later life, and occurs

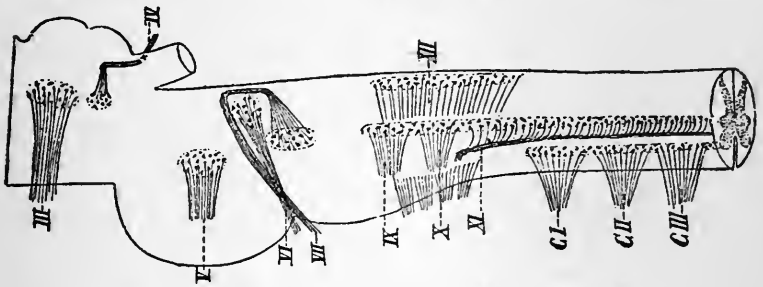


Fig. 62.—Showing the Relative Position of the Nuclei of the Motor Cranial Nerves (Van Gehuchten).

oftener in women; there is usually a neuropathic constitution; it is not hereditary. The exciting causes are anxiety, shock, injury, and exposures. It often has a reflex cause, usually from irritation of some branch of the trigeminus or the cervico-brachial nerves; rarely

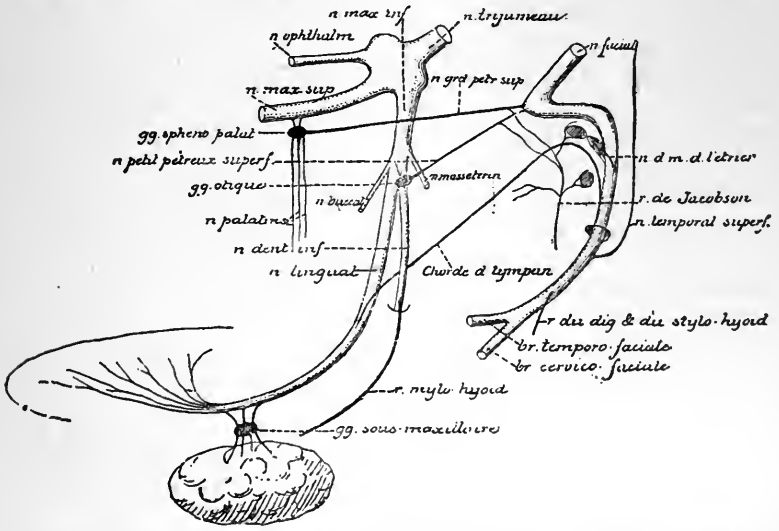


Fig. 63.—Showing the Course of the Taste Fibres in the Seventh and Fifth Nerves (Krause).

from eye strain. It is sometimes associated with tic douloureux. Organic diseases, such as tumors and softening, affecting the nerve nucleus in the pons or the cerebral centres, cause a symptomatic tic, but not the true disease. Thus we may have a post-hemiplegic tic

or a tic due to cortical lesion and associated with epilepsy. True facial tic may also be caused by irritation or disease of the nerve or its nucleus.

Symptoms.—The disease usually begins slowly and the orbicularis muscle and zygomatici are earliest affected. It rarely goes above the eyebrows, *i.e.*, to the corrugator supercilii and frontalis. The lower branch of the facial is little affected. The spasm is a clonic one; the muscles of the face are affected by a series of lightning-like twitches, with intervals of rest. Sometimes, however, the contraction becomes tonic and lasts several seconds or more. There is no pain. The spasm is increased by emotions, nervous excitement, conversation, exposure to light and cold, and is at its worst when the patient himself is most depressed. It is a very good gauge of the general nervous stability of the patient. There is no paralysis or atrophy, and there are no secretory or trophic symptoms. The taste fibres are rarely involved, though occasional subjective sensations of taste have been felt. The electrical irritability is either unchanged or slightly increased.

Associated movements of the eyes, of the jaw muscles and cheek muscles are sometimes seen. Pressure over the motor points of the nerve will sometimes arrest the movements for a time. The disease is a unilateral one. It lasts for years and even for the lifetime.

Pathology.—There is no known anatomical change in idiopathic cases. The disease is allied in character to wryneck and other chronic tics, and is a motor correlative to the severe neuralgias like tic douloureux. In these cases it is probably the expression of some local cortical degeneration of light grade. The disease is sometimes a pure reflex neurosis from ocular or dental irritations.

Diagnosis.—Idiopathic facial spasm is chronic, unilateral, unaccompanied by pain or paralysis. It is distinguished from facial spasms of organic origin by the fact that the latter always have some other symptoms. Thus facial habit chorea is bilateral; spasm from cortical disease is attended by disturbance of consciousness and comes on in paroxysms; the spasm occurring after hemiplegia is usually tonic, and so is hysterical facial spasm.

Prognosis.—The disease is in most cases incurable, especially after it has lasted some time. If a reflex cause exist, the prognosis is better. Life is, however, never endangered by it.

Treatment.—The most important thing is attention to the general health, removal of all depressing influences, rest, and freedom from excitement. Among specific remedies arsenic, the bromides, cannabis indica, gelsemium, conium, hyoscyamus, strychnine, codeine, and morphine are recommended. Morphine is useful, but must be

tried carefully and in small doses. Conium lessens the spasm, but this drug has to be given in large doses and is not entirely free from danger. Hyoscine and gelsemium sometimes do good. Careful examination of the teeth, eyes, nose, stomach, and uterus for reflex irritation is imperative.

Galvanism, if carefully and persistently applied, almost always helps. It should be given daily. Various methods are described. The best way is to place the negative pole on the sternum or back of the neck, and the positive pole over each motor point of the nerve for one to two minutes, then over the occiput and over the facial cortical area for the same time. Currents of from two to five milliamperes should be used. Neurectomy of the supraorbital, continuous pressure on the motor points, stretching the nerve itself, are all measures which may be considered valueless. Freezing the skin over the nerve with chloride of methyl has been recommended by Mitchell. Blistering and cauterization are needless inflictions. The anæsthetization of the conjunctiva with cocaine is often helpful, both in diagnosis and treatment.

Blepharospasm is the name given to a spasm of the orbicularis palpebrarum. It is generally caused by diseases of the eye, and its nature and treatment are matters belonging to ophthalmology. It is a rare symptom of hysteria.

Nictitating or winking spasm is a clonic spasm of the orbicularis, and usually forms part of habit chorea or is a symptom of hysteria.

Tonic facial spasm is sometimes seen in major hysteria.

Facial Palsies.

The paralyses of the facial nerve may be due to lesions that are central, nuclear, meningeal, or peripheral.

Facial palsy of *central origin* is almost invariably an accompaniment of hemiplegia and is due to hemorrhage, softening, inflammation, or tumor of the brain. The lower two branches of the facial are chiefly involved.

Facial palsy of *nuclear origin* is very rare and is an accompaniment of glosso-labial palsy, of diphtheritic palsy, or of gross lesions of the pons.

Facial palsy of *meningeal origin* is due to tumors, meningitis, or fracture of the base of the brain, and is accompanied by lesion of other cranial nerves. Syphilis is the most important factor here. All these forms are simply part of other diseases.

Peripheral facial palsy (Bell's palsy) is the common type of facial paralysis.

Etiology.—The typical cases of this disease are due to exposure, infection, and so-called rheumatic influences. Males are oftener affected, and the common age is between twenty and forty. It is more frequent in the winter and in temperate climates. It is not hereditary. A neuropathic tendency predisposes to it. Syphilis rarely causes an isolated facial palsy; in fact, it is apt to leave this nerve alone (Hutchinson). Facial palsy may occur in multiple neuritis, when it is often bilateral, and in locomotor ataxia. Non-typical and accidental cases of peripheral facial palsy are due to injuries, fracture of the petrous bone, or ear disease. Forceps pressure in difficult labor causes some cases, and a very few have been congenital.

Symptoms.—The disease comes on rather suddenly, and reaches its height within a few hours, or, at most, two or three days. Preceding and accompanying the onset there may be some pain about the ears and a little swelling is sometimes seen.

The patient feels a subjective discomfort on the paralyzed side of the face. He finds that he cannot completely shut the eye; if he tries to chew on the affected side, food gets between the teeth and cheek. He cannot pucker the lips, and his speech is a little muffled. The appearance of the face is most characteristic.

On the affected side the wrinkles are smoothed out, the angle of the mouth is lower, the mouth is drawn at first to the sound side, and owing to this distortion the tongue appears not to be protruded straight. In laughing or other emotional movements of the face, the trouble is most clearly brought out. But the most characterized appearance is produced by telling the patient to shut the eyes tightly and draw out the angles of the mouth so as to show the teeth (Fig. 64). The eye on the palsied side is not closed, and the eyeball turns up, showing the white of the eye. This test of the palsy is better than any examination of the wrinkles and folds of the face, for in children and in the young and plump these differences in the two sides of the face are not very marked, especially in the slighter cases. The nostril on the affected side does not expand on forced inspiration; the eye is apt to be watery and the conjunctiva somewhat injected.

If the disease extend well up into the Fallopian canal, so as to involve the nerve to the stapedius, that muscle is paralyzed, the tensor tympani acts unopposed, the drum is tightened, and unusual sensitiveness to sounds results. This is rare, however; most of the ear symptoms in facial palsy being due to a concomitant disorder of the tympanum or the acoustic nerve.

If the disease involve the nerve between the geniculate ganglion

and the point where the chorda tympani is given off (see Fig. 63), some loss of taste follows, and this is a frequent symptom, but not one of long duration. If the disease is located more centrally than the geniculate ganglion or more peripherally than the chorda nerve, taste is not involved. Usually, when there is no taste involvement, it is because the lesion is peripheral. By an examination of the

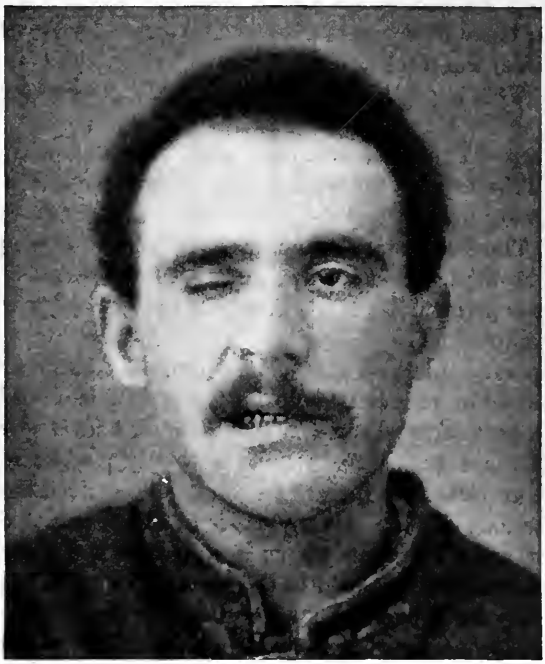


FIG. 64.—PATIENT WITH LEFT FACIAL PALSY ATTEMPTING TO CLOSE BOTH EYES AND SHOW THE TEETH.

taste sense and of the hearing, the location of the trouble can be accurately made out.

After a few weeks some wasting of the face may be noted, but this is never very great.

The electrical reactions are characteristic and important, since typical or partial degeneration reactions can usually be observed. For a few days there is an increase in irritability of the muscles to both faradic and galvanic currents. This is followed by a gradual loss or diminution in faradic irritability, while the galvanic irritability remains sometimes increased for a while and then falls. At the same time a reversal of poles and a sluggish contraction, particularly the latter, may be noted. At the end of five or six weeks faradic irritability ought to begin to return. Great variations are

found in the electrical reactions dependent upon the severity of the case. For example, in very severe cases the electrical irritability may be almost or entirely absent for days and even weeks.

If the disease lasts for two months or more and the palsy is not a complete one, secondary contractures begin to appear. The mouth now becomes drawn to the affected side, and the nasolabial fold becomes deeper than that on the sound side. In smiling or speaking or other facial movements there is an excess of movement on the paralyzed side, the teeth and upper gums in particular showing abnormally. This is particularly the case in old palsies occurring in childhood.

Pathology.—The disease in its typical form is due to a diffuse neuritis. This attacks the periphery of the nerve in the face and extends rapidly up into the Fallopian canal as far as the geniculate ganglion. The inflammation sometimes attacks most the peripheral filaments; at other times it is more central. The old idea that it was always a perineuritis of the nerve in the Fallopian canal is incorrect (Minkowski).

Facial paralysis is not, as is usually taught, a rheumatic disease. It is not caused by rheumatic poisoning, nor does it occur in persons who have a particular rheumatic constitution, and it is not in the majority of cases associated with a distinctly rheumatic etiology. It is rather an infectious disease and should be classed as such or as a post-infectious disease. Many cases will be found to occur after influenza or some infection which is allied to it. Indeed, it has seemed to me more like a cold of the seventh nerve than a rheumatism. This view is borne out by my experience in the treatment which shows that antirheumatic drugs, like the salicylates and iodides, do not appreciably modify its course.

Diagnosis.—The recognition of the palsy is made easy by causing the patient to contort the face. In children it requires more care to detect the side affected.

It is important to determine whether the palsy is cerebral, nuclear, basilar, or peripheral. If the cause is cerebral, the upper branch of the nerve is little affected and the *patient can close the eye.* The nerve and muscles show no degenerative reactions.

Nuclear palsy is very rare and is accompanied with other symptoms, especially those of involvement of other cranial nerves. A history of diphtheria, lead palsy, or bulbar paralysis is obtained.

In palsies due to lesions at the base of the brain, such as gummy meningitis, the auditory and other cranial nerves are involved and there are signs of brain syphilis. By testing the sense of taste and hearing, the location of the peripheral trouble can be made out.

Thus if there be loss of taste on the anterior two-thirds of the tongue, the lesion must be between the geniculate ganglion and the point at which the chorda tympani is given off. If the taste be not involved, the lesion must be central or peripheral of the part of the nerve which includes the chorda. Practically in most cases it is peripheral to it. If central the disease is usually of syphilitic or tuberculous origin; the palsy is severe and the loss of ability to close the eye very great.

Prognosis.—The prognosis of peripheral palsy (Bell's) is good, although an absolutely complete recovery often does not take place.

In syphilitic cases the prognosis is not so good, though patients may recover. In central palsies the prognosis is the worst, because the lesion usually does not disappear. However, the central cases are from the beginning of a mild type, and give annoyance mainly from the secondary contractures.

Bell's palsy usually lasts three to five months. Occasionally there are mild cases that get well within a month. The prognosis as to duration is much helped by a close study of the electrical reactions. In proportion as the degeneration reaction is complete and persistent the outlook is bad.

Treatment.—In the acute peripheral cases the patient should be treated promptly and thoroughly. He should be given a diuretic and purgative and a blister should be placed over the exit of the nerve. This should be followed or accompanied by hot fomentations. Salicylate of soda in full doses of twenty grains should be given during the first week. After the paralysis is established, iodide of potassium is indicated in moderate doses. Electricity is to be employed with care at first. After a week it may be given daily for five minutes, using the galvanic currents just strong enough to contract the muscles. After three or four weeks, if the faradic current causes contraction, it may be used, otherwise the galvanic current is to be continued. At the end of a month an application every other day is sufficient. At the end of three weeks, if the paralysis is severe, the corner of the mouth should be drawn up by means of a bent hook, which is carried back and fastened behind the ear. The patient should wear this most of the daytime, but not at night. The object is to take off the strain caused by the pulling of the muscles on the sound side.

At the end of a couple of months, if secondary contractures set in, massage may be tried, and the patient should practise facial gymnastics daily. Acupuncture combined with weak faradic currents and local injections of strychnine may be tried in obstinate cases. If the eye cannot be closed it should be protected by a shade.

The Glosso-Pharyngeal Nerve.

Anatomy.—The glosso-pharyngeal nerve has motor, sensory, and visceral fibres. The nucleus of origin of the motor fibres is the nucleus ambiguus common to it and the vagus and accessory. The sensory fibres arise from two small ganglia lying on the root of the nerve, the *petrous* and *jugular*. The cells of these ganglia are unipolar. The neuraxon bifurcates, and sends its central filament along the nerve root into the medulla, to the gray nucleus, known usually as the nucleus of origin of the glosso-pharyngeal and lying close to the vagus nucleus (Fig. 65). Fibres also go to a tract of gray matter lying close to the solitary bundle and known as the

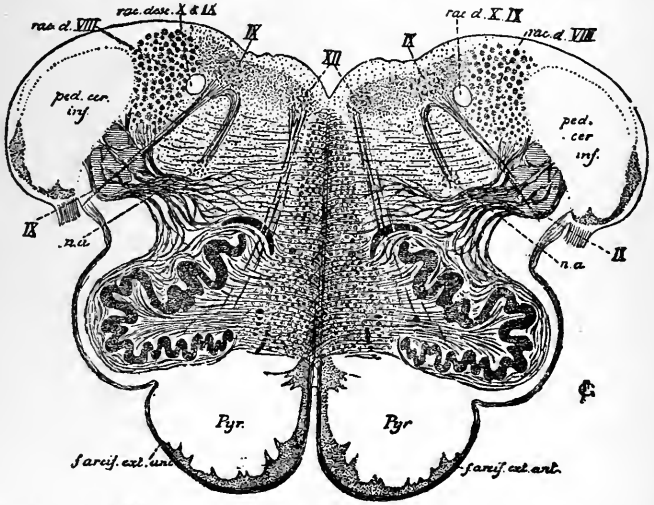


Fig. 65.—Section of Medulla, showing nuclei of origin of the *IX*, *X*, *XI*, and *XII* cranial nerves. *rac. d.*, Ascending sensory root of *IX*, *X*, and *XI* nerves.

ascending sensory root of the ninth, tenth, and eleventh nerves. The nucleus is really, however, a terminal one, and is the origin of secondary sensory neurons which send up fibres to the brain. The peripheral filament of the root ganglia passes along the nerve and supplies the fibres of sensation.

The nerve supplies general sensation to the tympanum, tonsils, and pharynx (in connection with the vagus) and upper part of the larynx; special sensation of taste to the posterior third of the tongue, and motion to the pharyngeal muscles and œsophagus (Kreidl) in connection with the vagus.

The terminal filaments of the sensory taste fibres supplying the posterior two-thirds of the tongue end in fine fibres that pass into the taste buds. There are no special peripheral cells of taste, as asserted by Fusari and Panasci.

Its cortical representation so far as taste is concerned is in the

hippocampal gyrus. The nerve gives very sensitive reflex fibres to the pharynx and is important in the reflex act of deglutition; it also carries sensations of nausea from pharyngeal irritation.

Motor Neuroses of the Glosso-Pharyngeal.

This nerve is rarely affected independently by motor troubles. Spasm of the pharyngeal constrictors occurs in general disorders like rabies, and reflexly in severe neuralgia of the trigeminus. This condition, known as *dysphagia*, is seen also in hysteria, and there is probably some spasm in connection with the symptom known as globus hystericus.

Paralysis of the throat constrictors occurs as one of the symptoms of glosso-labio-laryngeal paralysis and sometimes in diphtheritic paralyses.

Fig. 66.—The Vagus and Spinal Accessory Nerve (Young).

The Pneumogastric Nerve and the Accessory Part of the Spinal Accessory.

Anatomy. — The pneumogastric or vagus nerve has two nuclei of origin, a motor and a sensory.

1. The motor nucleus or nucleus ambiguus, which is a prolongation of the lateral horn of the spinal cord, lies deep in the medulla and is a nucleus common to the vagus and glosso-pharyngeal (Fig. 65).

2. The sensory fibres arise chiefly from two ganglia that lie on the root of the nerve, the jugular and plexiform. These bodies resemble spinal ganglia. The cells are unipolar and send off a neuraxon which bifurcates. The peripheral fibre passes along the nerve and supplies it with its sensory fibres. The central part passes up to the gray matter of the floor of the fourth ventricle and ends in the so-called sensory nucleus (Fig. 65).

This nucleus, however, is not the real nucleus of origin, but, as in the case of the ninth it contains cells which send their axis cylinders or neuraxons brainward and form secondary sensory neurons. Fibres also go to the ascending sensory root common to this nerve and the ninth. It is probable that some of the cells of the terminal sensory nuclei of the ninth and tenth are efferent visceral cells, like those of the columns of Clark, and send out visceral fibres. Both motor and sensory fibres are chiefly visceral in distribution and function.

The SPINAL ACCESSORY nerve is purely motor in function (Van Gehuchten). The accessory part rises from the cells of the nucleus ambiguus and passes into the trunk of the vagus.

The spinal part of the spinal accessory arises from the lateral horn and outer part of the anterior horn of the spinal cord. Its fibres of origin reach from the first to the third or fourth cervical nerves as far as the fourth or fifth cervical roots. The fibres unite in the cranium and pass out through the posterior lacerated foramen

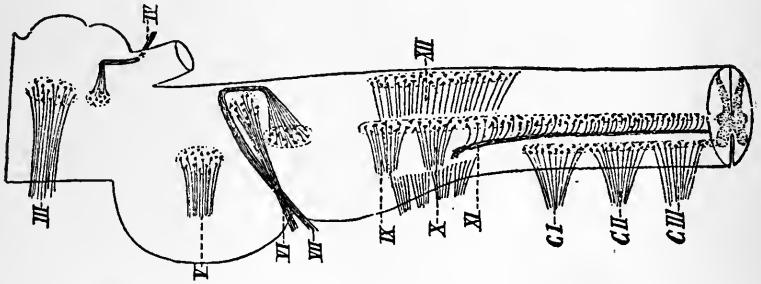


FIG. 67.—LONGITUDINAL SECTION SHOWING THE RELATIVE POSITION OF THE CRANIAL NERVE NUCLEI (Edinger).

in the same sheath as the vagus. After their exit from the skull, they divide into an internal and external part. It is the external branch which contains the fibres of spinal origin. The internal part contains the fibres from the medulla and unites with the vagus. The nerve receives some sensory fibres from the first and sometimes from the second cervical nerve. The terminal branches receive motor fibres from several cervical nerves. The spinal accessory contains large and small or visceral fibres. The spinal part contains only the large fibres.

The spinal accessory supplies the sterno-cleido-mastoid almost exclusively, but only the upper part of the trapezius; the rest of this muscle is supplied by the cervical and dorsal nerves. The sterno-cleido-mastoid, when innervated, draws the chin up and over toward the opposite side. The upper fibres of the trapezius draw the head back slightly and down toward the same side. Physiologically the spinal part of the accessory nerve is one of the motor cervical nerves; the accessory or medullary portion is part of the vagus, and has visceral and sensory as well as motor functions.*

* Dees thinks that the spinal origin of the eleventh is continuous above

The vagus and accessory part of the eleventh together have an extraordinary wide distribution and diversity of function.

1. First they contain motor, inhibitory, and vasomotor fibres. These fibres go to the pharynx, larynx, trachea, and bronchi; to the œsophagus, stomach, small intestines, and spleen.

2. Sensory fibres, which go to the occipital and transverse sinuses and dura mater of the posterior fossa, to the external auditory meatus in part, to the pharynx, larynx, and trachea, and to the œsophagus.

3. Excito-reflex fibres, which go to the lungs and heart, stomach, and to other organs mentioned as supplied by the vagus with sensation.

These reflex fibres stimulate or inhibit the vasomotor centre, the respiratory rhythm, and the cardiac rhythm. They also excite reflexly deglutition and respiratory movements.

The secretory fibres go to the respiratory tract, œsophagus, stomach, and pancreas and small intestines.

Cardio-inhibitory fibres go to the heart, while reflex accelerating fibres and inhibiting fibres go to the lungs. The accessory nucleus supplies the laryngeal adductors and the cardio-inhibitory fibres.

Diseases of the Pneumogastric Nerve and of the Bulbar Portion of the Spinal Accessory.

These nerves are essentially visceral in character. Their diseases call for a study of laryngeal, pulmonary, cardiac, and abdominal neuroses, which would bring us into the domain of laryngology and general medicine. Hence, despite their great importance, I have thought it best not to try and present them here. Some of the symptoms are described in connection with locomotor ataxia, progressive muscular atrophy, exophthalmic goitre, and angina pectoris.

Neuroses of the Spinal Part of the Accessorius.

This is a purely motor nerve, and its disorders are therefore spasm and paralysis.

Torticollis (Wryneck, Caput Obstipum).—Torticollis is a disease characterized by clonic or tonic spasm of the muscles supplied by the spinal accessory and often of other muscles of the neck. There are several forms of wryneck, which must be distinguished from each other. They are: 1, congenital wryneck; 2,

with the twelfth, not with the anterior nucleus of the tenth. He denies that the medullary nucleus sends fibres to the larynx.

The internal branch of the eleventh sends motor fibres to the rectus posticus (E. Remak).

symptomatic wryneck; 3, spurious wryneck, from spinal disease; 4, true spasmodic wryneck.

1. *Congenital wryneck* is due to some intra-uterine atrophy or obstetrical injury of the sterno-cleido-mastoid. It occurs oftenest after breech or foot presentations. The right side is usually affected. There is no spasm at all, but the neck is fixed to one side by the shortness of the muscle, and also rotated to the opposite side. The deformity becomes more noticeable as the child grows older, because the parts atrophy. The atrophy affects not only the shortened muscles, but the face on the affected side (Fig. 68).

2. *Symptomatic wryneck* is usually due to a rheumatic myositis, and occurs chiefly in children. It may be due also to tumors,

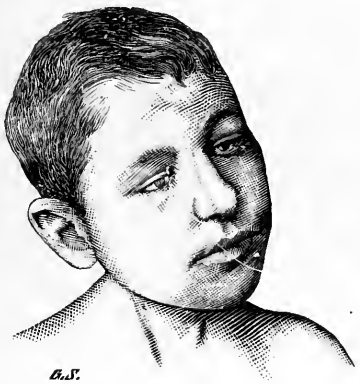


Fig. 68.—Congenital Wryneck of the Right Side.

adenitis, abscesses, and local syphilitic disease. In these cases there are always pain and tenderness associated with the deformity.

3. *Spurious wryneck* is an apparent or real spasm of the neck muscles due to caries of the spine.

Treatment.—Congenital wryneck, if taken early, can be cured by tenotomy of the sterno-mastoid and subsequent fixation of the neck for a time. When osseous changes have occurred perfect relief is impossible. Symptomatic rheumatic wryneck is a trivial and temporary affection, which needs only to be palliated by hot applications and saline purges until cure takes place.

Spurious wryneck requires suitable orthopædic measures, such as the plaster jacket and jurymast.

4. *Spasmodic Wryneck.*—This is a purely nervous disease characterized by spasm of the muscles supplied by the spinal accessory and often of those supplied by the upper cervical nerves also.

Etiology.—Women are much oftener affected than men. It

occurs in early adult and middle life, never in children or old people. A neuropathic constitution and heredity often exist.

The exciting causes are occupations which put the lateral muscles of the neck on a strain, depressing emotions, physical shocks and blows, rheumatic influences, and perhaps malaria. Sometimes no cause can be detected. Reflex irritations, perhaps, exist in some cases, but it is difficult to find them.

Symptoms.—The disease begins with slightly painful sensations in the neck, which are soon accompanied by spasm. The spasm is at first clonic and intermittent. The sterno-mastoid is oftenest involved of single muscles; but the rule is that the upper fibres of the trapezius are also affected. The patient's head is inclined toward

Fig. 69.—Typical Wryneck Involving the Spinal Acessory on the Right Side (Walton).

the affected side by the trapezius, the chin is raised, and the head rotated to the opposite side by the sterno-mastoid and trapezius, and this is the typical position in the disease (Fig. 69). If both trapezii are affected the head is pulled back, but this is a rare form. It is called retro-collic spasm. The complexus and obliquus superior are the only other neck muscles which can rotate the head to the opposite side. They are supplied by the upper cervical nerves and are sometimes involved in wryneck. In torticollis the muscles affected with spasm have a similar physiological function. Hence while the sterno-mastoid, trapezius, complexus, and superior oblique on one side are attacked by the spasms, muscles on the other side may be at the same time implicated. The opposite muscle commonly affected is the splenius, which inclines the head laterally and rotates it to the same side. Probably the deep muscles, recti capitis

postici, major and minor, and the inferior oblique, which draw the head back and rotate to the same side, are also at times affected. The list of muscles that may be involved and their nerve supply are as follows:

	Turning Head to Opposite Side.	Turning or Inclining Head to Same Side.	Nerve Supply.
Muscles usually involved.	Sterno-cleido-mastoid...	.....	Eleventh.
	Upper fibres of trapezius	.....	Eleventh.
Muscles rarely involved.	Superior obliquus.....	Recti capitis postici, maj., min., infer. obliq., splenius.	Cervical.
	Complexus		Cervical.

Extreme rotation without much retraction of the head would indicate involvement of the sterno-cleido-mastoid and opposite splenius. Retraction of the head indicates involvement of both trapezii.

The disease may start in one muscle and gradually extend to others, even involving the facial, masticatory, and brachial nerves. As it progresses the spasm becomes more constant, and finally it may be tonic, never yielding except to artificial means or during sleep. The pain associated with the disease gradually decreases. The affected muscles hypertrophy, the muscles thrown into disuse atrophy. There is some deformity, in time, of the neck and shoulders, but facial asymmetry does not occur in this form unless it begins, as is very rarely the case, before maturity.

The disease may be complicated with or alternate with other neuroses. I have known epilepsy to be associated with it.

Pathology.—The disease is a neurosis involving the bulbar and cerebral centres. The neuro-mechanism controlling the movements of the neck is unstable and out of control. Consequently it sends out intermittent and irregular discharges of nerve force. The seat of the typical disease is never in the nerve alone, probably the cortical centre controlling the mechanism of the head movements is at fault. The disease is a degenerative one, and indicates the premature decay of centres never perhaps originally perfect.

The *diagnosis* has to be made from the other forms of wryneck mentioned. The age, history, and fixed character of the spasm serve to distinguish congenital wryneck. The history, the pain and tenderness, and the temporary duration differentiate the rheumatic forms.

The increased rigidity on passive motion, the pain, deformity, and other signs of cervical caries are sufficient to diagnosticate vertebral disease.

Prognosis.—The disease is not fatal. It generally reaches a

certain stage and then remains chronic. In rare cases it is cured; in many others it can be much ameliorated. Cases occurring in young people, in the hysterical, and in those without a decided neurotic history are the most favorable.

Treatment.—The drugs which are efficient are opium, atropine or hyoscyamine, conium, gelsemium, valerianate of zinc, asafœtida, chloral, bromides, arsenic, and cocaine. Of these, opium, atropine, gelsemium, and zinc are generally the most efficacious. Opium must be given with great caution. Atropine should be given hypodermically in increasing doses up to intoxication (gr. $\frac{1}{10}$) (Leszynsky). The galvanic and faradic currents are useful adjuvants in helping to relax the spasm and keep up the nutrition of the muscles; but alone they are not curative. Massage and stretching the neck in a Sayre apparatus, together with systematic exercise of the neck muscles, often help; neck stretching will even sometimes cure. The only surgical measures to be advised are nerve resection, and possibly the partial cutting of the sterno-mastoid muscle. A very few cures and many failures have followed surgical interference.

Splints and mechanical-fixation apparatus do no good as a rule.

Resection of the posterior branches of the upper three or four cervical nerves, as suggested by Keen, has done great good in a few cases (Powers). Prolonged rest for eight to twelve months with massage and exercises is on the whole the most satisfactory remedy

Spasmus Nutans (*eclampsia nutans, nodding spasm, salaam spasm, oscillating spasm*).—This is a disorder occurring chiefly in children and characterized by rhythmical nodding or oscillating movements of the head.

Etiology.—The disease occurs in young children who are anæmic and badly nourished. Dentition, digestive disorders, basilar meningitis, gross disease of the brain, are causes. Sometimes it is only a kind of habit chorea, and occasionally this habit continues during life.

Symptoms.—The disease may come on suddenly; more rarely it develops slowly. The patient has paroxysms usually of a rather violent character, lasting for minutes or hours, or even continuing nearly all the time except during sleep. The head moves thirty to sixty times a minute usually, but the motion may be slower or faster. Movements of the eyes and facial muscles often complicate the affection. The paroxysm may end in an epileptic attack.

The *diagnosis* is easily made by the symptoms. The *prognosis* and *treatment* depend upon the etiology. I have found useful bromide of potassium, hyoscine, and syrup of iodide of iron.

Paralysis of the Spinal Part of the Accessory—*Etiology.*—The causes are injuries, caries of vertebra, progressive mus-

cular atrophy, and all forms of spinal disease reaching high up in the cervical cord.

Symptoms.—When one nerve is paralyzed the head may still be held straight, but there is inability to rotate it perfectly. The prominence of the sterno-mastoid is absent—atrophy takes place. No spasm of the other muscle occurs, and there is no such thing as paralytic torticollis (Gowers). The involvement of the trapezius causes a depression in the contour of the neck, especially noticeable on deep inspiration. There is some trouble in raising the arm, the scapula is drawn away from the spine, and the lower angle is rotated inward. When both nerves are paralyzed there is great difficulty in rotating the head or raising the chin. Paralysis of both sterno-mastoids causes the chin to drop backward, while paralysis of both trapezii in their upper parts causes the head to drop forward. Atrophy of the muscles attends the paralysis of the nerve, and degenerative reactions may be noted. The cervical nerves appear sometimes to supply the sterno-mastoid and upper part of the trapezius so much that in disease of the accessories decided paralytic symptoms are absent. When both parts of the spinal accessory are involved, dropping of the palate, dysphonia, and rapid pulse are added symptoms.

The *diagnosis* depends upon a thorough examination of the motility of the parts.

The *treatment* is based on a knowledge of the cause of the disease.

The Hypoglossus—XII.

Anatomy.—The hypoglossal nerve arises from a long and large nucleus lying in the lower part of the floor of the medulla near the median line and to the outer and ventral side of the central canal. The nucleus is a continuation upward of the anterior horns of the spinal cord and is homologous with the sixth, fourth, and third nerve nuclei higher up (see Fig. 67). It reaches below as far as the decussation of the pyramids and above as far as the glosso-pharyngeal nucleus. A second small-celled nucleus lies just beneath the nucleus proper. Its cortical representation is in the lower end of the central convolutions, to which it is connected by fibres that pass into the raphe and thence to the anterior pyramids. Its fibres pass out between the olivary body and the anterior pyramid. At its origin it is a purely motor nerve; it receives a few sensory fibres from the cervical nerves and the vagus. It supplies the following muscles:

Intrinsic muscles of the tongue: superior and inferior longitudinal and transverse. The extrinsic muscles of the tongue: hyoglossus, genio-hyoglossus, and styloglossus. (The palato-glossus and linguals are supplied by the fifth and seventh cranial nerves

respectively). The depressors of the hyoid: the thyro-hyoid and, with the cervical nerves, the sterno-hyoid and sterno-thyroid. The elevator of the hyoid: genio-hyoid. It is also thought to send fibres to the oral muscles (Tooth).

The hypoglossal nerve is concerned in the movements of the tongue and in fixing or depressing the hyoid in mastication and deglutition. When diseased, therefore, speech and deglutition are affected. The small nucleus of the nerve is thought to control the finer lingual movements of articulation.

Motor Neuroses of the Hypoglossal Nerve.

The diseases of this nerve consist of lingual spasms, lingual palsy, and lingual hemiatrophy.

Lingual spasms take part in the disorders of articulation, helping to cause stuttering and speech cramps. Such troubles are often developmental in origin and belong to the habit choreas or convulsive tics.

Stuttering is a spasmodic disorder in which the tongue muscles are involved, preventing the proper enunciation of words and sentences.

Stammering is an imperfect articulation due sometimes to disease or defect in the hypoglossal nerve and its muscles. It is not a spasm.

The letters that are oftenest badly pronounced are R, L, and S. Lisping is a form of stuttering. Stammerers are sometimes called the "R L S people."

Aphthongia is the name given to a form of spasm occurring in speakers and similar in nature to writer's cramp.

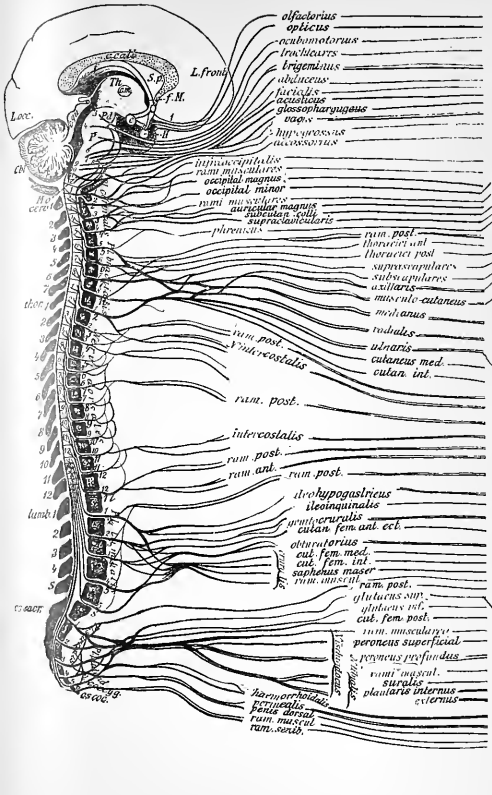
Clonic lingual spasm occurs in chorea, hysteria, and during the attacks of epilepsy. Unique cases of this spasm also occur from reflex irritation or central nervous disease.

Tonic lingual spasm occurs in hysteria, and sometimes as an independent affection due to unknown causes, generally those of a debilitating and nervously depressing character. Reflex irritation may be a cause.

Lingual paralysis (glossoplegia) is usually one of the symptoms of glosso-labio-laryngeal palsy. It may be caused by a bilateral or even a single lesion in the cerebral hemispheres. The condition is then known as pseudo-bulbar paralysis. Diseases of the medulla and of the nerve itself may cause the paralysis.

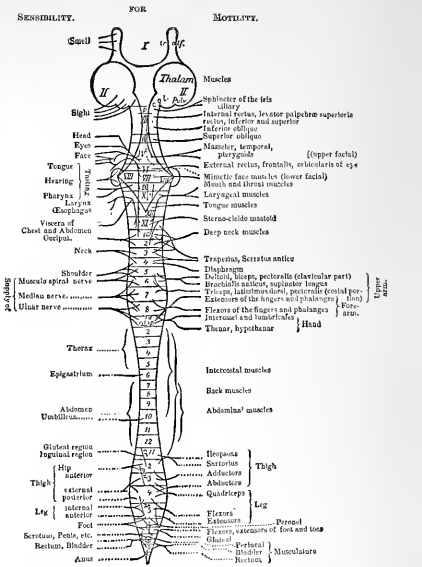
The paralysis may be either unilateral or bilateral. The symptoms are an impairment of speech and of swallowing. Fuller details will be given under the head of Bulbar Palsy.

Progressive Lingual Hemiatrophy.—A progressive hemiatrophy of the tongue sometimes occurs. It is analogous in all respects to facial hemiatrophy, with which it is sometimes associated. It is probably due to a low grade of degenerative neuritis of the nerve. It is very rare.



1. (a) interior eye muscles (intercalation of the ciliary ganglion), ciliary muscle for accommodation—sphincter of the pupil for contraction of the pupil.
   (b) exterior eye muscles: levator palpebræ superioris; rectus superior, internal, inferior—
2. Superior oblique of the eye (movement downward and outward).
3. Muscles of mastication: masseter, temporal, pterygoid, mylo-hyoid—ant. digastric, tensor tympani, tensor and levator veli palati. Secretion of tears?
4. External rectus of the eye (outward movement).
5. The mimic face muscles: frontal, orbicular of the eye and mouth; zygomatici, mentalis; platysma. Of the ears, stapedius, posterior digastric, etc. Nasal secretions. Tear secretion? Palate musculature?
6. Throat musculature (constrictors), crico-thyroid, secretion of gastric juice. Movements of
7. Tongue musculature, sterno-hyoid, thyreoideï. [heart and breathing.
8. Larynx musculature (voice formation), palate, pharyngeal musculature (with the vagus (act of swallowing)—sterno-cleido-mastoid (drawing the head laterally and nodding). Trapezius (in part) (drawing the shoulders). [head).
9. Posterior superficial and deep head and neck muscles (elevating and lateral movements of
10. Deep posterior and anterior back muscles. Trapezius in part (movement of head and throat).
11. Scaleni (elevators of the ribs, breathing), longus colli.
12. Diaphragm (breathing).
13. Deep neck muscles.
14. Pectoralis major (anterior adduction of the upper arm), pectoralis minor, subclavius.
15. Levator scapulæ, rhomboids (dorsal nerves of the scapula) (draws the scapula inward and upward), serratus anticus major (long thoracic nerve) fixes the shoulder blade and draws the same in conjunction with the arm above the horizontal.
16. Supraspinatus (raises and rolls the arm outward), infraspinatus, teres minor (rolls outward).
17. 8 subscapularis, teres major (rotates inward), latissimus dorsi (adducts the arm and draws it
18. Deltoid (raises the arm to horizontal). [backward).
19. Biceps (flexor of forearm and supinator), brachialis anticus (flexor of forearm), coracobrachialis.
20. Flexor communis digitorum (radial portion) (bends the terminal phalanges), flexor longus pollicis (end phalanx), flexor carpi radialis, pronator radii teres, and quadratus, palmaris longus, thenar muscles (opponents of thumb flex the primary phalanges and extend the terminal phalanges), lumbricales 1, 2, seldom 3 (flex the peripheral phalanges and extend simultaneously the terminal phalanges).
21. Triceps (extensor of forearm), radial portion of brachialis anticus, supinator longus (flexes and pronates the forearm), extensor communis digitorum (extensor of the peripheral phalanges), extensor pollicis—abductor pollicis, supinator brevis, extensor carpi radialis and ulnaris.
22. Flexor profundus digitorum (ulnar portion), see 26. Flexor carpi ulnaris, hypothenar, interosset (flex the peripheral phalanx), lumbricales (3 and 4), see 20, adductor pollicis.
23. Deep back muscles.
24. Intercostal muscles.
25. Deep back muscles (extensor of trunk).
26. Intercostal muscles.
27. Back muscles.
28. Intercostal muscles, abdominal muscles (rectus, external oblique) (compressor of abdomen).
29. Back (lumbar) muscles.
30. Transverse abdominal, internal oblique (compressor of abdomen).
31. Cremaster, transverse, oblique.
32. External obturator, adductor of thigh, gracilis (adductor).
33. Psoas (lumbar plexus), iliacus internus (draws up the thigh, flexes the trunk), quadriceps (extends the leg), sartorius.
34. Gluteus medius, minimus (abductors of thigh), tensor vaginæ femoris, pyriformis, obturator internus (abductors).
35. Gluteus maximus (extends the thigh).
36. Gemelli; pectineus, semitendinosus, semimembranosus (flex the leg), quadriceps femoris (adductor).
37. Tibialis anticus (raises the inner border of foot), peroneï (raise outer border of foot), extensor digiti communis.
38. Gastrocnemius, soleus (plantar flexion of the foot), flexor digiti, tibialis posticus.
39. 40. Small muscles of foot (flexor brevis, interossei, etc.).
41. Levator, internal sphincter ani, sphincter vesicæ.
42. Sphincter ani, perineal musculature, bulbi cavernosi, etc.
43. External sphincter ani.

LOCATION OF THE SEGMENT



Explanation of the Abbreviations.—*tr. olf* = olfactory tract; *c.g.l.* = lateral geniculate body; *p., r., cr., pat., A.*, represent approximately the location of the reflex centres; the pupillary reflex (*p.*), for the respiratory reflex (*r.*), for the cremaster reflex (*cr.*), patellar reflex (*pat.*), and the Achilles reflex (*A.*). The centres for the bladder and rectum in the sacral cord are represented by circles; likewise the centres for erection and ejaculation. The centre for contraction of the uterus is probably likewise in this vicinity (Jakob).

CHAPTER IX.

NEUROSES OF THE MOTOR SPINAL NERVES.

Anatomy and Physiology.—The spinal nerves arise from the spinal cord by two roots, anterior and posterior. These roots unite outside the spinal canal to form mixed nerves. The mixed nerves divide and go to their various destinations. There are thirty-three pair of spinal nerves, viz.:

Cervical	8	
Dorsal	12	
Lumbar	5	
Sacral	5	
Coccygeal	3	(all rudimentary).
	33	

The last two coccygeal nerves are microscopic in size, and the first pair is very small, so that practically there are but thirty sets of spinal nerves.

The posterior roots are closely connected with ganglia lying in the intervertebral canal, and called intervertebral ganglia, or ganglia of the posterior roots. These ganglia are the real origin of the great majority of the fibres of these roots. The mixed nerve is connected, by fibres that come chiefly from the anterior root, with the sympathetic or prævertebral ganglia. The distribution of the spinal nerves is also shown in Fig. 70.

For the purpose of conveniently studying the diseases of the spinal nerves, we divide them into six different groups, each having a somewhat definite work to do. These groups are shown in the accompanying table.

	Strands of Spinal Nerves.	Distribution.	Associated Ganglia of Sympathetic.
Group I.	Upper four cervical.	Occipital region, neck.	First cervical.
" II.	Lower four cervical and first dorsal.	Upper extremities.....	Second and third cervical, first dorsal.
" III.	Upper six dorsal.	Thoracic wall.	First to sixth dorsal.
" IV.	Lower six dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.	Fifth to twelfth dorsal.
" V.	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.	First to fourth lumbar.
" VI.	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg.	First to fifth sacral.

Group I. The Upper Cervical

includes the first four of the spinal nerves. These divide into anterior and posterior branches. The posterior branches supply the

muscles and skin of the back of the neck and the occiput. The principal nerves are the suboccipital and the great occipital. The anterior branches form the cervical plexus. Its principal branches are the auricularis magnus, occipitalis minor, and phrenic. The

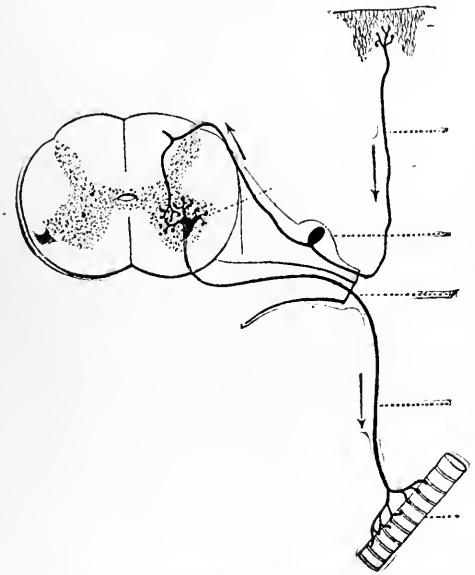


Fig. 71.—Showing the Formation of the Mixed Nerves and the Mode of Origin of the Motor and Sensory Parts.

special distribution of the nerves is shown in the table facing Fig. 70.

The upper cervical nerves supply motion to the muscles which rotate the head and draw it back and sideways.

One branch, the phrenic, supplies the diaphragm; other muscles assist in fixing the thorax in forced inspiration. They innervate some of the hyoid and thyroid muscles, but have no influence on phonation or deglutition. This group of nerves is in close connection centrally with the trigeminal nerve, whose descending root reaches down into the cervical cord. The fibres to the scalp and face also anastomose with the trigeminus in their peripheral distribution to the scalp and chin.

The diseases of the upper cervical group are spasms, paralyses, and neuralgias.

Spasmodic Diseases.—Torticollis may be limited to the cervical nerves, as has been shown.

Tonic spasm causing a rigid neck is a frequent symptom of meningitis, and forms part of epileptic and other convulsions.

In oscillatory and rotatory spasms the cervical nerves are involved.

In some forms of STUTTERING the phrenic nerve is involved in a clonic or tonic spasm.

HICCOUGH is a clonic spasmodic disorder of the phrenic nerve. It is usually due to gastric disturbance, with flatulent distention of the stomach.

When chronic, it is caused by hysteria, neuritis, diaphragmatic pleurisy, or some pressure upon the nerve in its course. I have seen cases in which it was probably a pure spasmodic neurosis. Ordinarily, hiccough can be stopped by simple carminatives like spirits of chloroform or lavender. In obstinate cases in which no known cause can be found, pilocarpine, hyoscine, and bromides are useful. Counter-irritation along the neck helps some cases. A most effective measure is to lay the patient supine over a thick bolster so that the head hangs back and the thorax arches up. An injection of morphine and atropine promptly stops some cases.

PARALYSES AND ATROPHY.—The cervical muscles are paralyzed in progressive muscular atrophy, in pachymeningitis hypertrophica, and occasionally in vertebral and pheripheral disease or injury. Some deformities and weakness in head movements result, but the most serious consequence is involvement of respiration through palsy of the phrenic.

PARALYSIS OF THE PHRENIC NERVE—*Etiology.*—Such paralysis may be due to disease or injury of the cervical cord and also to peripheral disease, to which the nerve is somewhat liable owing to its long course through the anterior mediastinum.

Pleurisy, peritonitis, mediastinal tumors, rheumatic and toxic influences, and hysteria are among the special causes of phrenic paralysis. Spinal-cord disease, such as tabes, acute ascending paralysis, and surgical injuries are, however, the commonest etiological factors.

Symptoms.—In diaphragmatic paralysis, if bilateral, as is usually the case, there is either no movement of the abdomen or the epigastrium and hypochondrium are drawn in. On slight exertion there are dyspnœa and increase of respiration.

Diagnosis.—If no other muscles than the diaphragm are involved, the cause is probably in the trunk of the nerve. Inflammatory disease of the diaphragm may cause a paralysis which is recognized by its painful character and the febrile reaction.

Treatment.—This is to be guided by the cause. It need only be said that there is a motor point in the neck where by careful electrization one can get a contraction of the diaphragm (see Fig. 36, *B*). In paralysis of the phrenic this fact should be borne in mind.

Group II. The Lower Cervical Nerves and Brachial Plexus.

Anatomy and Physiology.—The anterior branches of the lower four cervical nerves and first dorsal nerve unite to form the brachial

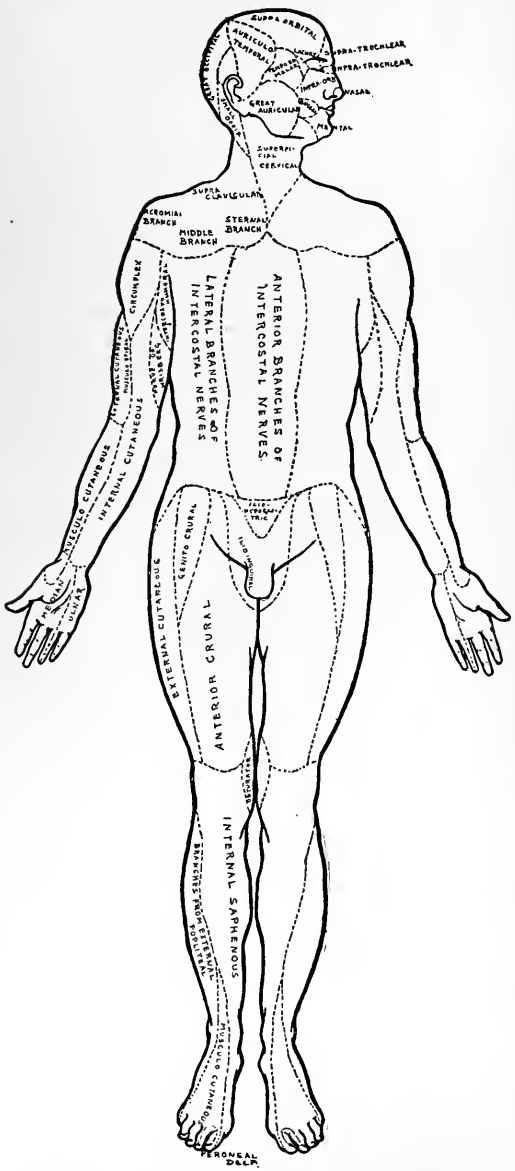


Fig. 72.—Showing the Distribution of the Sensory Nerves of the Skin.

plexus. This gives off *short nerves* to the shoulder and trunk and *long nerves* to the arm.

The mode of formation of the brachial plexus is shown in the diagram (Fig. 73). It is in accordance with the descriptions of Walsh and Allen. The *short* or upper branches supply the shoulder and intercostal muscles. The *long* or lower branches supply the arm and hand. The neurologist needs to know: (1) the muscular distribution of each nerve and the function of the muscle; (2) the cutaneous sensory distribution; and (3) the level of origin of the nerves.

The previous figure, Fig. 70, and table give these points, and will be found useful for study and reference. They are based upon the investigations of Ferrier and Yeo, Thorburn, and also on Abbe's and my own experiments.

The Arrangement of the Brachial Plexus.—It is made up of three nerve trunks, which in turn make up three cords, these cords giving off various branches, thus:

1. Trunk from sixth and seventh cervical roots } forms outer cord, which gives off { Ext. ant. thoracic. / Musculo-cutaneous. / Outer head median.
2. Trunk from eighth cervical and first dorsal roots } forms inner cord, which gives off { Inner head median. / Ulnar. / Int. cutan. / Int. ant. thorac. / Intercost.-hum.
3. Trunk from fifth, sixth, seventh, and eighth cervical and first dorsal } forms posterior cord, which gives off { Subscapular. / Circumflex. / Musculo-spiral.

The following table shows the origin, muscular distribution, and effect of paralysis on the motor but not on the sensory nerves. This latter is indicated in Figs. 72 and 32.

Nerves and Roots of Origin.	Muscular Distribution.	Function as Shown by Effect of Paralysis.
Posterior thoracic. Fifth and sixth cervical.	Serratus magnus.	Posterior edge of scapula is rotated out when arm is raised and carried forward. Weakening of elevation of shoulder and of inspiration.
Circumflex. Fifth, sixth, seventh and eighth cervical.	Deltoid. Teres minor.	Loss of power to raise arm.
Suprascapular.	Supraspinatus.	Weakened power to raise arm; head of humerus tends to fall. Loss of abductors, motion forward and inward rotation of humerus.
	Infraspinatus and teres minor.	Loss of rotation of humerus outward.
Subscapular, short. Fifth and sixth cervical.	Subscapulares. Teres major.	Weakens inward rotation of arm. Weakens power of elevating shoulder.
Subscapular, long. Fifth, sixth, seventh, and eighth cervical.	Latiss. dorsi.	Weakens power to depress shoulder and to pull arm backward and to side.
Anterior thoracic.	Pectoralis major.	Loss of power to pull arm down and forward and to shrug the shoulder.
Musculo-cutaneous. Fourth and fifth cervical.	Biceps and brachialis anticus.	Loss of flexion of forearm and weakness of inspiration.
Musculo-spiral.	Triceps.	Loss of extension of forearm.

Nerves and Roots of Origin.	Muscular Distribution	Function as Shown by Effect of Paralysis.
Fourth, fifth, sixth, seventh, and eighth cervical.	Supinatores.	Loss of supination.
	Extensor carp. rad. Extensor carp. uln.	Extension of wrist lost except when fingers are flexed; lateral deviation.
	Extensor comm. digit.	Impaired extension of first phalanges and wrist.
	Extensor p. i. pollic. Extensor s. i. pollic. Extensor oss. met. pollic.	Impairment of extension of thumb.
Median.	Pronatores.	Loss of pronation.
Fifth, sixth, seventh, and eighth cervical.	Flexor carp. rad.	Weakened flexion of wrist.
	Flexor sublim. dig.	Weakened flexion of second and third phalanges of first and second fingers.
	Flexor profund. dig. radial half.	
	Two lumbricales.	
	Abductor pollic.	Loss of abduction and flexion of thumb.
	Flexor pollic.	
Ulnar.	Flexor carp. ulnar.	Weakened flexion of wrist; radial deviation.
Eighth cervical, first dorsal.	Flexor profund. dig. ulnar half.	Weakened flexion of second and third phalanges of third and fourth fingers.
	Interossei.	Loss of flexion of first phalanges and of extension of second and third.
	Two lumbricales.	
	Flexor minor digit.	
	Abductor pollicis.	
	Inner half of flexor brev. pollicis.	

Diseases of the Lower Cervical Nerve Group and the Brachial Plexus.—The nerves of this group are subject to the pathological disturbances common to all nerves. I shall describe them from the clinical side, which gives the following disorders:

- Paralyses
  - Combined arm palsies.
  - Upper-arm type or Erb's palsy.
  - Lower-arm type.
  - Paralysis of individual nerves.
- Spasmodic Disorders.
  - Occupation neuroses.
- Sensory Disorders.
  - Brachial neuralgia.
  - Digital neuralgia.
  - Numb hands.
- Secretory, Trophic, and Vasomotor Disorders.
  - Neurotic œdema.

Spasmodic Disorders of the Arm.—The arms and hands are especially subject to tremors, choreic and other spasmodic movements. The only spasmodic disorders, however, which may be said to be especially located there are writer's cramp and allied occupation neuroses. These are described elsewhere.

Brachial Paralyses, Arm Palsies.—These occur as combined or total-arm palsies, upper-arm and lower-arm types, and paralyses of single nerves.

Combined paralysis of the brachial nerves is a condition in

which all or nearly all the portions of the plexus and its branches are involved. Total arm palsies make up about six per cent. of all peripheral paralyses, and are about one-fifth as frequent as single-nerve paralyses.

Etiology.—They occur oftenest in men, but are not rare in infants, being then due to injuries during parturition. After infancy they are most frequent in early and middle life.

The exciting causes are obstetrical and other injuries; deep-seated inflammations and tumors; shoulder dislocations; primary neuritis; crutch and other forms of mechanical compression; func-

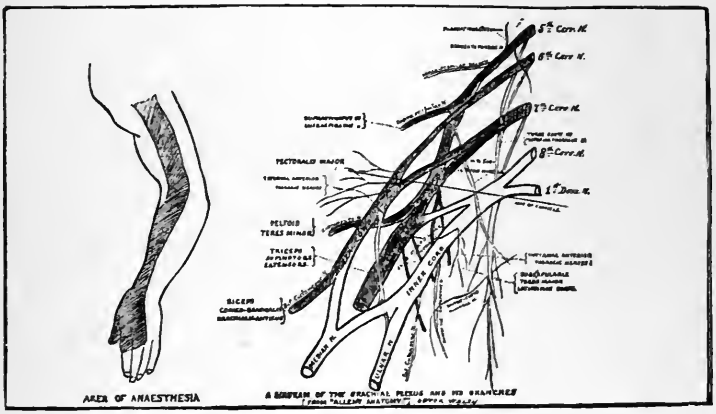


FIG. 73.—ILLUSTRATING THE FORMATION OF THE BRACHIAL PLEXUS; ALSO THE INVOLVEMENT OF THE PLEXUS IN DEGENERATIVE NEURITIS (LESZYNSKY).

tional palsies, from overwork and hysteria; in rare cases, spinal-cord and brain disease.

The *symptoms* vary with the severity and extent of the lesion.

With regard to severity, there are three degrees. In the first there is simply a transient palsy, due to lying too long on the arm. The arm feels heavy, numb, and "asleep." In a few minutes or hours this palsy disappears. In the second degree the nerves are so much compressed as to be mechanically injured. If the patient has been drinking hard, even moderate pressure may set up an inflammatory or destructive process that leads to quite a serious palsy. In the third degree the nerves are actually cut or torn across, or so compressed as to lose their anatomical integrity.

The resulting symptoms are those common to all nerve injuries, viz., paralysis, wasting, changes in electrical reaction of the muscles. Pain, tenderness, anæsthesia, trophic, secretory, and vasomotor disturbances are also present in varying degree.

The distribution of the paralysis, atrophy, and sensory disturb-

ance depends, of course, upon the arm nerves chiefly involved. The cuts and table will enable one to see in any case where the trouble is localized.

There are three common symptoms, however, of which it is often very important to analyze the cause. These are the loss of power for elevation of the arm and for flexion and extension of the forearm.

Flexion of the forearm is performed by the biceps and brachialis anticus, and is helped by the supinator longus. These muscles are

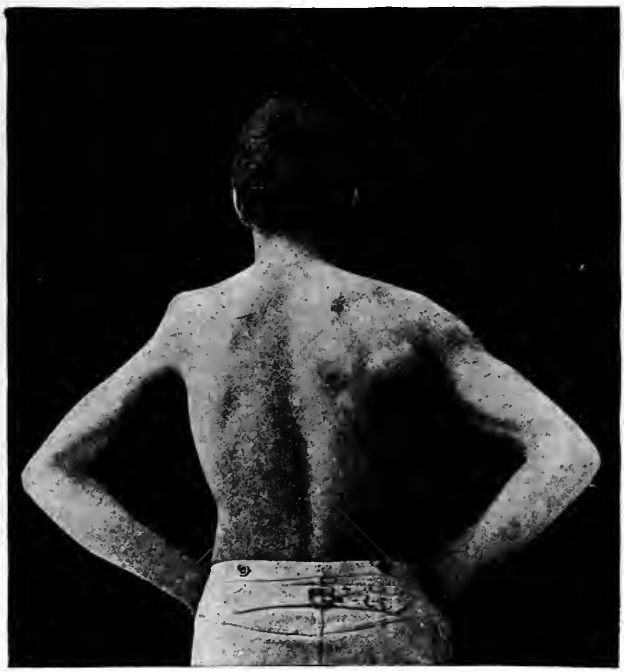


FIG. 74.—UPPER-ARM PALSY OF LEFT SIDE.

supplied by the musculo-cutaneous nerve, except the supinator, which is supplied by the musculo-spiral. Hence when a person cannot flex the forearm, the musculo-cutaneous is chiefly affected.

Extension of the arm is done by the triceps, which is supplied by the musculo-spiral.

Elevation of the Arm Outward.—Inability to raise the arm is the common and striking symptom in combined brachial palsies. The arm is raised by a number of muscles. The deltoid acts first and most, but it can raise the arm only to a right angle. It is supplied by the circumflex nerve from the posterior cord of the plexus. After the arm is raised to a right angle, it is further elevated by

rotating the scapula, and this is done chiefly by the middle part of the trapezius (lower cervical and upper dorsal nerves) and by the serratus magnus, supplied by the posterior thoracic nerve. A number of other muscles combine to strengthen elevation of the shoulder, but this action can be abolished only by paralysis of the deltoid or trapezius and serratus mangus.

The *diagnosis* of these cases involves, first, the consideration of the seat of the lesion and the special nerves involved; next, that of the nature of the lesion. A recognition of the seat of the lesion and of the special nerves involved depends entirely upon the study

FIG. 75.—LOWER-ARM PALSY DUE TO MULTIPLE NEURITIS.

of the distribution of the palsy and of the atrophy and sensory disturbance. It is important to determine whether one is dealing with a total-arm palsy, an upper-arm type (Erb's palsy) or a lower-arm type.

In *Erb's palsy* there is involvement of the deltoid, biceps, brachialis anticus, and supinator longus, with at times paralysis of the supinator brevis, and even of all the muscles supplied by the median nerve. The lesion is either in the cord formed by the fifth and sixth cervical nerves or a little lower in the brachial plexus, where the fibres supplying the musculo-spiral, circumflex, and musculo-cutaneous lie close together. At all events, the lesion involves the central parts and upper cords of the plexus. The arm hangs by the side and the forearm cannot be flexed (Fig. 74).

In the *lower-arm type* the triceps, the flexors of the wrist, the

pronators, the flexors and extensors of the fingers, and the hand muscles are involved. The arm can be raised and the forearm flexed and supinated, but the hand is useless and the extension of the forearm is impossible. The lesion here involves chiefly the nerves from the seventh and eighth cervical and first dorsal roots (Fig. 75).

If the lesion is in the nerve there will be atrophy, changes in electrical reaction, sensory disturbances, and often, if there is neuritis, pain over the nerves. The reflexes will be lessened or absent. If the lesion is in the spinal cord, symptoms in other parts of the body will be present, or, if not, there will be no sensory disturbance, as in an arm palsy from anterior poliomyelitis. In rare cases arm palsies may be caused by spinal tumors or a local meningitis, in which case diagnosis is difficult.

The *upper-arm type* and *lower-arm type* palsies are caused by much the same factors as the combined palsies; their symptoms have been indicated above. The upper-arm type is especially frequent in infants and constitutes one of the obstetrical paralyses. It may in some cases be due to injury or hemorrhage in the cord.

The *prognosis* is usually good. Nearly all these cases get well, the duration of the incapacity being from two or three months to a year. Even in the severest cases recovery is possible after one or two years. If, however, the nerves are torn across and the ends widely separated, recovery is doubtful unless an operation is promptly done.

The *treatment* consists in electrical applications, mechanical support, with potassium iodide internally and abstinence from alcohol. Local injections of nitrate of strychnine are useful, and massage should be used if it can be applied carefully.

In brachial palsies due to severe injuries, dislocations, fractures, etc., in which there is evidence, from the extreme atrophy and absence of electrical reaction, that the nerve is entirely cut across and that the ends are not in apposition, a surgical operation is stringently needed. The nerves should be exposed and the ends brought as near together as possible. Decalcified-bone tubes or sterilized macaroni may be used to give a passage for the central end to grow into the peripheral. In these cases, however, it must be remembered that the two ends do not unite; but the central end grows down in the tract of the old degenerated peripheral stem.

A peculiar form of combined nerve palsy sometimes occurs, due apparently to a *primary brachial neuritis*. It begins in the plexus and involves first the nerves of the upper cervical roots. It may extend down and involve the ulnar, median, or musculo-spiral.

It occurs in adult men generally and in those exposed to rheumatic influences. Perhaps lead-poisoning may exist.

It begins gradually with slight pains and weakness in the shoulder and arm muscles. Atrophy and anæsthesia follow, and degeneration reactions are present. There is not a great deal of pain (Fig. 74). The disease is usually confined to one side. It lasts several months, and nearly complete recovery finally takes place. Relapses may occur.

It is differentiated from progressive muscular atrophy by the pain, anæsthesia, and electrical reactions, and from arthritic atrophy by the absence of any history of arthritis and the presence of degeneration reactions and anæsthetic areas.

Paralyses of Single Nerves—Paralysis of the Posterior Thoracic Nerve—*Etiology.*—This rare trouble usually occurs in male adults and is due to injury or sudden strains. Its paralysis may be part of a progressive muscular atrophy. The nerve goes to the serratus magnus.

Symptoms.—When paralyzed, there is difficulty in raising the arm above the horizontal position and the movements of the shoulder are weakened. When the arm hangs by the side the lower angle of the scapula is drawn a little nearer the vertebral column and protrudes slightly. When the arm is held out horizontally the inner edge of the scapula protrudes and is drawn toward the middle line. When the raised arm is brought forward there is a deep groove formed between the inner border of the scapula and the thoracic wall (Fig. 76.)

The disease often runs a long course and is accompanied by pain.

Paralysis of the Circumflex Nerve.—The nerve goes to the deltoid, teres minor, third head of the triceps, and shoulder-joint. It gives sensation to the skin of the shoulder. It is very often paralyzed. The commonest causes are a fall or injury, dislocation, and rheumatic inflammation of the joint. The arm cannot be elevated or rotated outward. There are atrophy, anæsthesia, and sometimes pain.

Paralysis of the Suprascapular Nerve.—The nerve goes to the spinati muscles, teres minor, and shoulder-joint. Disease of this nerve alone is rare.

The supraspinatus rotates the shoulder in, the infraspinatus and teres minor rotate it out. When paralyzed, there is an impairment of rotation and some impairment of elevation of the shoulder.

Paralysis of the Musculo-Spiral Nerve (*Wristdrop, Lead Palsy, Compression Paralysis*).—The distribution of this nerve is

given in the table and cut. Its function is to extend and supinate the forearm, to extend the wrist and fingers, and to adduct and abduct the fingers slightly. It extends directly only the last or un-

FIG. 76.—PARALYSIS OF SERRATUS MAGNUS OF LEFT SIDE (Leszynsky)

gual phalanges, the first and second phalanges being extended by the ulnar nerve.

Etiology.—The musculo-spiral, owing partly to its course, is the most frequently affected by paralysis of all the arm nerves. Pressure on the nerve during sleep, especially when the patient is intoxi-

cated, crutch pressure, fractures, wounds, tumors, lead poisoning, arsenical, alcoholic, and other forms of multiple neuritis are the causes of its disordered function.

Symptoms.—The symptoms of this paralysis are "wristdrop," due to an inability to extend the wrist or fingers. The first and second phalanges can be extended somewhat by the interossei and lumbricales, but the last phalanges cannot be extended at all. The first finger is least affected. The fingers can be only slightly abducted, supination is generally lessened or lost; if the lesion is high up, the triceps is involved and the power of extending the forearm

Fig. 77.—Paralysis of Musculo-Spiral Nerve and Wristdrop.

weakened. There may be atrophy of the muscles and degeneration reaction. A swelling over the tendons of the wrist-joint may occur. Some numbness and tingling exist, and occasionally there is anæsthesia in the distribution of the radial nerve on the hand. The disease lasts but a few weeks if due to compression; for months if due to neuritis, lead poisoning, or severe injury of the nerve. Eventually recovery takes place.

When the disease is due to *lead poisoning* there are some peculiarities in its course. Thus the supinator longus usually escapes; the palsy begins gradually and usually involves both arms; it may extend to the upper arm. Partial degeneration reactions are present. There is rarely any anæsthesia and but little pain. Often there is a lead line on the gum and a history of constipation and colic.

In *alcoholic* and other forms of multiple neuritis there are pain and paræsthesia, both arms are involved, and the flexors and other forearm muscles are somewhat implicated. There are marked sensory disturbances. The legs are also affected.

In *compression palsy* the supinators and often the triceps are involved.

When the lesion of the nerve is high up, as in *crutch paralysis*,

there is but little anæsthesia, and that is found on the anterior surface of the forearm, in the distribution of the external and internal cutaneous nerves. Lesion of the nerve lower down may give rise to some anæsthesia along the radial border of the forearm and back of hand; but the anæsthetic area varies a great deal.

The *diagnosis* of the paralysis is easily made. The most important point is to find out the cause. The different characteristics of lead palsy, neuritic palsy, and compression palsy have been indicated in the description of the symptoms. One must be sure to exclude also progressive muscular atrophy.

The *treatment* consists of mechanical measures such as electricity, massage, the application of rubber muscles, and in bad cases the fixation of the forearm and hand in hyperextension by means of a splint and plaster-of-Paris bandage (Gibney). Internally in the early stage it is best to administer iodide of potassium and sulphate of magnesium (in lead palsy), the salicylates or nitrate of silver in neuritis; later, hypodermic injections of strychnine may be given. Static sparks, galvanism, and other forms of electricity unquestionably do good in some cases, as I have had opportunity to prove.

Paralysis of the median nerve is rare as an isolated trouble, and is almost always due to injury or neighboring lesions. Embolism of the axillary artery after labor has produced it.

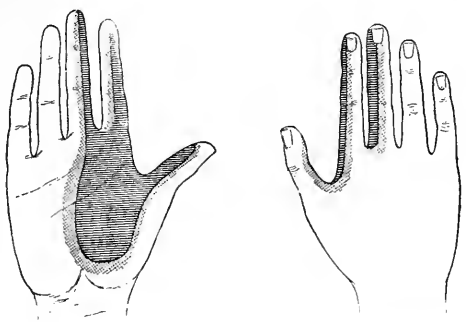


Fig. 78.—Area of Anæsthesia in Paralysis of the Median Nerve.

When paralysis occurs the grip is weakened. Flexion and abduction of the thumb and flexion of the first and second fingers are impaired. Atrophy of the thenar eminence may occur. The anæsthetic area varies, but is well shown in the accompanying cut (Fig. 78).

Paralysis of the Ulnar Nerve—*Etiology.*—The ulnar nerve is rather commonly affected by paralysis, the occurrence rank-

ing next in frequency to musculo-spiral palsy. It is rarely affected in lead poisoning, but is usually early involved in progressive muscular atrophy. It is sometimes attacked by a primary degenerative neuritis. Injuries are the common cause.

The *symptoms* are shown by the table (p. 227). The hand cannot be closed tightly, the little and ring fingers being especially weak. The first phalanges are drawn back and the second and third phalanges flexed; when the interossei and lumbricales atrophy, the result is the "griffin claw" or *main en griffe*. The fingers cannot be adducted or abducted except feebly.

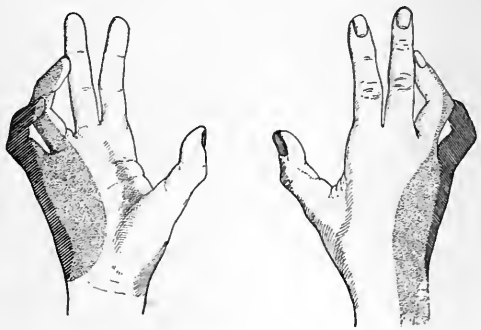


Fig. 79.—Showing Area of Anæsthesia in Ulnar-Nerve Palsy (Bowlby).

There is anæsthesia over the area of distribution of the ulnar (Fig. 79); there may be pain and tenderness.

Symmetrical Spontaneous Ulnar Neuritis.—A form of neuritis of the ulnar nerve sometimes occurs which has certain peculiar characters. It develops slowly in persons with a neuropathic history, and without known exciting cause. There are first some pain and paræsthesia in the region of the ulnar nerve of one hand; this is followed by weakness and atrophy of the mucles supplied by the ulnar nerve, and characteristic deformity appears. Anæsthesia develops coincidently. The other hand soon becomes affected. The disease is very chronic, and complete recovery is rare. It is probably a form of degenerative neuritis, and is due to some infection.

Migrating neuritis is a serious but rare malady which deserves some mention. It occurs as a sequel to some wound of or operation upon a nerve. The local neuritis extends usually up the arm (ascending neuritis). It is accompanied by intense pain, anæsthesia, paralysis, and atrophy. The disease is very chronic and intractable. It has been relieved in some cases by resecting the posterior spinal roots.

Morvan's Disease, Analgesic Paralysis with Whitlow.—(Neuritic type of syringo-myelia.)

This is a very rare disease, characterized by a slowly progressive paralysis and atrophy of the hands and forearms, with analgesia and painless whitlows.

It occurs usually in young or middle-aged males. Occupations, such as handling fish, which involve exposure and trauma, predispose to the disease.

The symptoms come on slowly, with, at first, severe pains in the arms and hands. The muscles of these parts gradually get

Fig. 80.—Showing the Hands in a Case of Paralysis with Whitlows (Erb).

weak and atrophy. Anæsthesia and analgesia are present. Usually there is loss of pain and temperature, but not of tactile sense. Whitlows appear which are painless, and one or more of the terminal phalanges may necrose. There is usually a swelling and hard œdema of the parts. Both upper extremities may be affected, and sometimes the feet are also slightly involved (Fig. 80). There is sometimes spinal curvature. Hysteria may complicate the affection. The course of the disease is very chronic, but there may be long periods in which the symptoms are quiescent.

Pathological examinations always show a neuritis of the parts involved in the disease. In some cases a syringo-myelia has been found. There is, therefore, a Morvan's disease due to syringomyelia and neuritis, and another type due to a neuritis alone.

The prognosis, so far as cure is concerned, is bad, but the disease may remain stationary a long time or improve.

The diagnosis is based on the combination of paralysis, atrophy, loss of pain and temperature but not of tactile sense, and whitlows. Leprosy must be excluded.

Strychnine seems to have stopped the progress of the malady in one case. The treatment in general is only symptomatic.

Group III. The Thoracic or Dorsal Nerves.

Anatomy and Physiology.—The dorsal nerves are twelve in number. The first is the largest and belongs functionally to the arm nerves. The dorsal nerves carry motor and sensory fibres to the voluntary muscles, skin, and other tissues of the trunk wall. They also carry splanchnic fibres to the lungs and abominal viscera. They divide into anterior and posterior branches. The anterior form the *intercostal nerves*, of which the first six are distributed to the wall of the thorax and the last six to the wall of the abdomen. All these nerves give off lateral and anterior branches. The posterior branches of the dorsal nerves are small, and supply the muscles and skin of the back.

Besides the above, there are recurrent branches which enter the spinal canal and supply the intraspinal veins and membranes. The first six dorsal nerves contain fibres which enter the sympathetic and supply the pleura and lungs. Fibres from the last six enter the sympathetic and supply the pelvic viscera. The intercostal nerves in their course lie part of the way beneath the pleura; they also pass along in close company with the intercostal artery and vein, lying just above these in the groove on the internal surface and inferior border of the ribs.

The upper six dorsal nerves, including both branches, are mainly inspiratory in function. They also extend and rotate the dorsal and cervical vertebræ. The lower dorsal are expiratory nerves; they also assist in compressing the abdominal viscera, and in flexing, extending, and rotating the spine.

Motor Neuroses.—The thoracic motor nerves are mainly involved in respiratory cramps and paralyses; sneezing, coughing, laughing, and crying are symptoms in which they play a large part. In complete respiratory paralysis also these nerves are affected. But there are few motor neuroses that are limited to the thoracic nerves. The neuroses of these parts are mainly sensory and will be described later.

Group IV. The Lumbar Nerves.

Anatomy and Physiology.—The lumbar nerves are five in number. The posterior branches supply the erector spinæ, interossei, multifidæ spinæ and interspinales, and also the skin of the back. The anterior branches of the upper four unite to form the *lumbar plexus.* The fifth or lumbo-sacral nerve sends most of its fibres to the sacral plexus. The branches of the lumbar plexus are: (1) the ilio-inguinal, (2) ilio-hypogastric (from first lumbar), (3) genito-crural, and (4) external cutaneous (mainly from the second), (5) obturator (from third and fourth lumbar), (6) the anterior crural (from second, third, and fourth—Fig. 81).

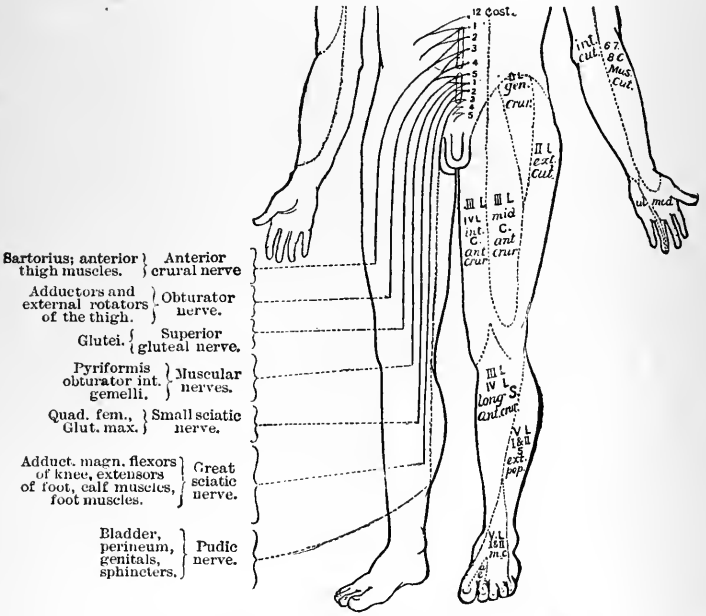


Fig. 81.—Diagram showing the Origin and Distribution of the Motor and Sensory Nerves of the Lower Limbs.

The first four branches of the plexus are comparatively short and supply sensation to the abdominal wall and external genitals. The last two are longer and supply the hip and knee joints, the muscles of the anterior, inner, and outer part of the thigh, the skin over this region and the inner side of the leg and dorsum of the foot (Fig. 81).

The diseases of the lumbar nerves and plexus so far as they form independent disorders are mainly the neuralgias. In making a diagnosis of lumbar-nerve disease, one should remember that of

the six branches of the plexus the upper four are mainly sensory, the lower two mixed nerves.

Paralyses and spasmodic troubles of the lumbar nerves are not rare, but are usually symptomatic of some extrinsic and often serious disease.

Etiology.—Hence it is well to catalogue here those affections which may produce lumbar palsies or spasms. They are pelvic tumors or injuries, impacted fæces, caries of the spine, psoas abscess, obturator hernia, hip disease, and pressure of the fœtal head.

Symptoms.—When the upper two lumbar nerves are involved, only sensory symptoms in the distribution of their branches occur (Fig. 81). If the next two are also involved, there may be trouble in extending the leg and flexing the hip on the trunk. The patient cannot raise himself to a sitting posture. If there are irritation and spasm, the thigh is drawn up and adducted.

In *paralysis of the obturator nerve* there are loss of power to adduct the thigh and cross the leg and weakness of outward rotation of the thigh. Anæsthesia over the inner side of the thigh may be present.

In *paralysis of the anterior crural nerve* there are weakness of the muscles of the anterior surface of the thigh, loss of power of extending the leg, and anæsthesia or pain over the crural area.

Paralysis of the posterior branches of the lumbar nerves causes weakness or paralysis of the erectors of the spine. The lumbar curve is very greatly exaggerated, the shoulders being thrown far back and the belly protruding. This condition occurs in progressive muscular atrophy, particularly in the pseudo-hypertrophic form.

Group V. The Sacral Nerves.

Anatomy and Physiology.—The sacral nerves are five in number. The first four divide into anterior and posterior branches. The fifth has no anterior branch. The posterior branches escape through the posterior sacral foramina and supply the multifidus spinæ and the skin over the sacrum and coccyx.

The first three anterior branches, with the lumbo-sacral nerve and a branch from the fourth sacral, unite to form the sacral plexus. This lies upon the pyriformis muscle in the pelvis, and escapes at the lower part of the sacro-sciatic foramen. The great mass of the fibres go to make up the sciatic nerve.

The roots of origin of the sacral and coccygeal nerves form the cauda equina.

The branches of the sacral plexus are the superior gluteal, muscular, small sciatic, inferior gluteal, pudic, great sciatic, perforating, cutaneous, and articular. These are distributed to the muscles, skin, and joints of the buttocks, thighs, legs, and feet. Their distribution is shown in Fig. 81.

The sacral nerves are the main agents in station and locomotion. They control the legs entirely, also the posterior muscles of the thigh and buttocks; they give sensation to these parts. They carry also fibres that regulate the sexual function, bladder, and rectum. From the sacral portion of the cord there is an outflow of nerves to the sympathetic, thence to the pelvic organs.

The diseases of the sacral nerves may be classified in a similar way to those of the brachial plexus.

Spasmodic Disorders of the Sacral Nerves.—Tremor, rigidity, clonic and tonic spasms, myoclonus, athetoid movements, all affect the lower extremities, but they are almost invariably part of some general or central disorder, such as chorea, paralysis agitans, hysteria, etc. Under the head of occupation neuroses there occur certain rare spasmodic troubles special to the legs. Saltatory spasm involves the legs alone. These disorders are, however, general neuroses.

Peripheral Leg Palsies.—Paralyses of the lower limbs from disease of the nerves may be either combined or single, just as is the case with arm palsies.

A combined sacral palsy is one in which all or nearly all of the branches of the sacral plexus are involved.

Etiology.—Such palsies are due to much the same causes as those affecting the lumbar nerves, viz., injury, dislocation, hip disease, tumors, and neuritis. Hysteria may cause a functional sacral palsy.

Symptoms.—The symptoms are indicated by a study of the distribution of the nerves, varying, however, in degree. The foot cannot be moved; the leg can be slightly extended by the anterior crural, but not flexed; the thigh cannot be drawn back freely or rotated perfectly. There is anæsthesia over the distribution of the sacral nerves; pain may be present; wasting and vasomotor and secretory disturbances occur unless the paralysis is functional.

The course depends on the severity of the lesion. If the nerve is totally cut or torn across, it may require one or two years for perfect recovery, which, however, occurs if the severed ends are properly approximated.

The *diagnosis* of a sacral palsy is based on the history and on the distribution of the anæsthesia and of the muscular paralysis. The sacral nerves do everything for the lower limb except extend the leg, flex and adduct the thigh, and to some extent rotate it. They supply sensation equally extensively (see Fig. 81).

The distinction from spinal-cord disease is chiefly based on the unilateral symptoms, the absence of disorder of the sphincters, and

the combination of paralysis, wasting, and sensory troubles, in the course of the sacral nerves.

Single-Nerve Sacral Palsies.—The symptoms of paralysis of single nerves are indicated by their function. The nerves rarely affected are the superior gluteal, muscular, and small sciatic. The nerve oftenest affected is the *great sciatic*, and especially its *anterior tibial branch*. In the latter case a condition called "dropfoot" is produced.

In the *pathology* and *treatment* of sacral palsies there is nothing especial that can be said.

CHAPTER X.

SENSORY NEUROSES OF THE CEREBRO-SPINAL NERVES.

As the most common disorders of the sensory nerves are neuralgias and paræsthesias, I shall introduce the subject with a general description of these symptoms.

PARÆSTHESIA.

Local Paræsthesia, Acro-paræsthesia, Waking Numbness.

The condition known as paræsthesia is one which should be more familiar to physicians, and be more commonly recognized and understood. *Paræsthesia* is the name given to a number of subjective sensations, such as prickling, numbness, creeping sensations, tickling, and burning. It includes, in fact, nearly all the subjective sensations of the skin, except those of pain. It is a condition which is, therefore, extremely common, and in its mildest and most trivial character is much more often experienced than pain. When these sensations fix themselves in a certain locality, following perhaps the tract of the nerve, or fastening themselves upon the hand or foot, they take on a certain clinical picture, and deserve to have the name of a disease, to just the same extent that a neuralgia does. Paræsthesia, in almost all cases, implies simply a lower grade of irritation of the nerve fibres than occurs in neuralgia, and is a kind of ghostly simulacrum of that disease. It very often precedes or accompanies attacks of pain. There is sometimes a tingling of the teeth or burning in the face which has a shadowy likeness to a toothache or trigeminal neuralgia. In the same way, one finds paræsthesias affecting the head, causing sensations of pressure and constriction, of burning, and general undefinable discomfort, which are entirely comparable to headaches.

In conditions of neurasthenia, paræsthesias of the head are more common even than the headaches. Paræsthesia sometimes follows the course of a nerve, as when one feels numbness of the hand if the ulnar is pressed upon at the elbow, or numbness in the foot when the sciatic is pressed upon, as when the legs are crossed.

There is also paræsthesia affecting one of the intercostal nerves or one of the crural nerves. On the other hand, paræsthesia may

affect all four extremities, so that they feel entirely benumbed or prickling. There is, I repeat, a very close analogy between these groups of paræsthesias and neuralgias.

Etiology.—Paræsthesia, whether local or diffuse, occurs rather more often in women than in men, and rather more often in the mature and middle-aged than in young people. It is most frequent in women of middle age, especially in those who are accustomed to hard work with the hands in washing and the exposure incidental to this. It also occurs in those who are obliged to walk a great deal and to be upon their feet, and it especially affects tailors, seamstresses, bookbinders, and those who have to use their hands constantly in some skilled mechanical work. It sometimes occurs in old age, being accompanied by evidence of gout or glycosuria in some cases. It is associated with rheumatism (twelve per cent of cases) and with alcoholism (eight per cent of cases). It follows infections like typhoid fever and the grippe, and means in these, as in many other cases, a low grade of neuritis. As a rule, however, paræsthesia is a neurosis in which exposure, rheumatism, alcohol, *arterial sclerosis*, and lithæmia are the causes.

Paræsthesia affects single cerebro-spinal nerves just as neuralgia does, or it may be more generally distributed. In the latter case it affects most the feet and hands, and it is called *acro-paræsthesia*.

We meet then with:

1. Cephalic paræsthesias, comparable to diffuse headaches.
2. Local paræsthesias, compararable to local neuralgias.
3. Acro-paræsthesia, involving the feet or hands or both diffusely.

The cephalic paræsthesias are usually symptoms of neurasthenic or lithæmic states and will not be considered here. Among eighty-five cases of local and acro-paræsthesiæ, not symptomatic of other and organic nerve disease, I found that there were of the local forms thirty-five cases, of acro-paræsthesia fifty cases. The local paræsthesias affected the arms in eighteen cases, next the thigh and leg nerves in twenty cases, and, last, the trigeminal nerve in three cases.*

* The following analysis of eighty-five cases of paræsthesia occurring in my practice shows something of the cause and local development of the malady. The most frequent causes I find to be those concerned with occupation. Parasthesia, in its general manifestations, may be considered almost an occupation neurosis. The list of cases may be put down as follows:

Occupation	15	Reflex irritation	2
Rheumatism	10	Hysteria	3
Alcoholism	6	Climacteric change	2
Infection	6	Various causes, such as neurasthenic state, puerperium, etc.	12
Senility	6		

Among 85 cases there were 36 males and 49 females:

Symptoms.—The patients have feelings of numbness, prickling, coldness, heaviness, usually in both hands, extending a little way up the forearm and often also in the feet. The sensations are, as a rule, diffuse, but may follow the distribution of a nerve, especially the ulnar. In other cases the numbness is sharply limited to the finger tips or the second and third phalanges. There is no actual pain or tenderness, nor is there any anæsthesia, cutaneous or muscular. Instead of or with the numbness there may be burning sensations, especially felt in the palms or soles. There is often a slight degree of paralysis, but rarely any decided vasomotor or trophic changes. The symptoms exacerbate, being worse at certain periods of the day, usually in the morning. They may increase at night and prevent sleep. The arms are oftener affected than the lower extremities; and the symptoms never except in hysteria take a hemiplegic form, though one arm may be alone involved. The scalp and ears may be involved, though always in a minor degree, the patient complaining of sensations of heat, prickling, flushing, and vertex pressure. In the local forms, there is numbness along the ulnar nerve or in one arm or along some branch of the lumbar nerves (*meralgia*). The symptoms cause a condition of mental unrest and nervousness which add to the sufferings materially. Evidences of overwork, of dyspepsia and constipation, of anæmia, or of muscular rheumatism will generally be found. The urine is usually rather light in color and specific gravity (1.010 to 1.018), slightly cloudy, with excess of phosphates and occasional excessive discharges of urates. Under the head of paræsthesic neurosis, the affection known as *waking numbness* or *night palsy* may be described.

This is a disorder characterized by a temporary paralysis of an extremity, with numbness, noticed on first waking or after lying down for a time.

	Males.	Females	Total.
Hands and feet, or both, affected	6	11	17
Hands alone	6	12	18
Feet and legs	10	10	20
General sensations		4	4
Local	14	12	26
	36	49	85

The special nerves affected were:

Trigeminal	4	Sacral	1
Brachial	5	Sciatic	1
Ulnar	7	Plantar	11
Radial	1		35
Crural	4		
Peroneal	1		

It is a rare disease and little is known of its cause. It occurs in adults and usually in the neuropathic. Sometimes evidence of weak heart or poor innervation of the vessels is present.

The symptoms are much like those caused by temporary compression of a nerve when the leg or arm goes to sleep. The paralysis is temporary and there is no anæsthesia. It is often a very obstinate condition but leads to no serious result.

Pathology.—The pathology of paræsthesia is based upon the similarity of the symptoms to those in mild or incipient cases of neuritis and upon the effects of drugs in relieving symptoms. It is believed that the peripheral nerves are being irritated by some poison circulating in the blood. Co-operating with this are nerves naturally over-irritable, or made so by anæmia, hyperæmia, or exposure to cold. In some cases there is undoubtedly a low grade of neuritis, and in other cases there is a congestion or slight degree of degeneration of the nerve.

Diagnosis.—The condition is to be distinguished from hysteria, neurasthenia, multiple neuritis, occupation neuroses, and central organic diseases of cord or brain. In hysteria the symptoms are less diffuse, regular, and bilateral, while some anæsthesia is rarely absent. Paræsthesias are very common in neurasthenia, and in some cases here they are due, no doubt, to peripheral irritation. In neurasthenia, the paræsthesias are, however, generally in one extremity, less diffuse, and as a rule more temporary. There is no motor weakness, and the head and spine are usually complained of more than the arms or legs. Waking numbness and night palsy are intermittent disorders, disappearing within a short time after waking or rising from a recumbent posture. The numbness that is felt with digiti mortui and Raynaud's disease is accompanied by spasm of the blood-vessels and whitening of the fingers. In multiple neuritis there is usually some distinct anæsthesia, pain, or tenderness, and also motor weakness.

Prognosis and Course.—The disease, if not treated, runs a course of several months, with some intermissions and relapses. It may disappear, to return the next year. It never progresses to any serious condition, and is in almost all cases eventually cured.

Treatment.—The patient should receive alkalies, with bromides, pepsin, iron, and strychnine. The faradic current, massage, and stimulating liniments are useful. Rest and removal from exciting causes, such as exposure and special work, are also indicated.

NEURALGIA.

Neuralgia is a condition characterized by pain in the course of a nerve or of nerves.

Forms.—Neuralgias may be idiopathic, *i.e.*, developed spontaneously; or symptomatic, *i.e.*, due to some known toxic influence, reflex irritant, or organic disease acting on the nerve. Thus a central disease of the nervous system or a tumor pressing on a nerve may cause a symptomatic neuralgia. When there is organic disease of the nerve itself, such as neuritis, the disease cannot be, strictly speaking, called neuralgia, but it is often impossible to draw the lines absolutely.

Neuralgias are divided, in accordance with their cause, into the epileptiform, hysterical, reflex, traumatic, gouty, etc.

Neuralgias are also divided, in accordance with their anatomical location, into trigeminal, cervico-occipital, brachial, intercostal, lumbar, crural, sciatic, and visceral.

Migraine and headache are not classed among neralgias.

Neuralgias of all kinds make up about ten per cent of the nervous disorders for which the neurologist is consulted. The most frequent form is the trigeminal; next in order come the sciatic, intercostal, cervico-occipital, lumbo-abdominal, brachial, and visceral neuralgias.

Etiology.—Neuralgia never affects young children, and, leaving out migraine and headache, it is rarely met with before the fifteenth year. From that time until twenty-five the frequency very rapidly increases. About one-fourth of all cases occur between the ages of fifteen and twenty-five; the relative number then slowly decreases to the forty-fifth year, when there is a rapid fall. Neuralgia is very rare in old age.

Women are more affected than men, in the proportion of five to three (analysis of 887 cases). In New York most cases occur in winter, next in the autumn. More cases occur in temperate climates and in wet and cold regions.

Hereditary influence, neurotic constitution, anæmia and debility, gouty and rheumatic diatheses, all predispose somewhat to the disease. The exciting causes can be included under the head of toxic agents, infections, exposure, overexertion, emotional shock, injuries, and neuritis of low degree.

Local irritation of nerves may excite neuralgia, direct or reflex in kind. Some help in recognizing the reflex origin of neuralgias and other pains may be gained from the accompanying diagrams.

Symptoms.—These will be described in detail in the chapters on the special forms.

The dominant symptom is, of course, pain. This pain is sharp, darting, boring, stabbing, or burning in character. It comes on in

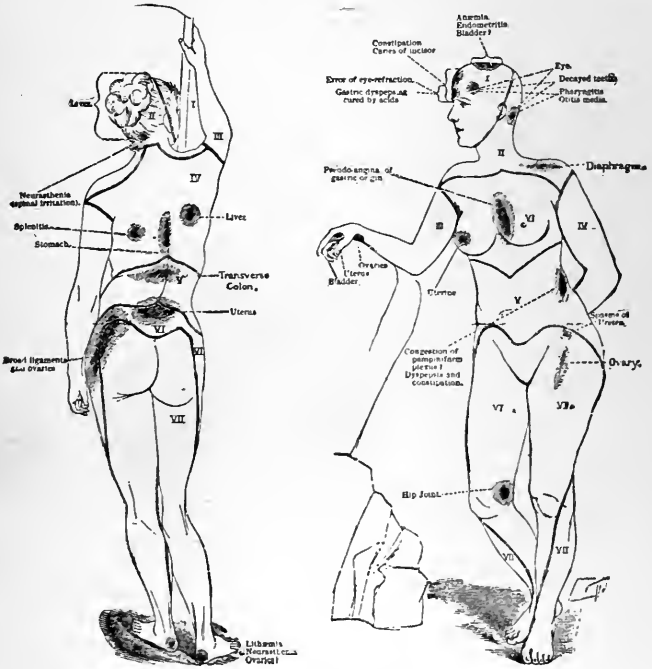


FIG. 82.—DIAGRAMS SHOWING THE DISTRIBUTION OF THE CEREBRO-SPINAL STRANDS OF NERVES AND THE LOCATION OF TRANSFERRED PAINS AND NEURALGIA.

	Strands of Cerebro-Spinal Nerves.	Distribution.
Area I.....	Trigeminus, facial, etc.	Face and its orifices, anterior scalp.
Area II.....	Upper four cervical.	Occipital region, neck.
Area III.....	Lower four cervical and first dorsal.	Upper extremities.
Area IV.....	Upper six dorsal.	Thoracic wall.
Area V.....	Lower six dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.
Area VI	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.
Area VII.....	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg.

paroxysms of great intensity. In the intervals there may be no pain or it may be simply a dull ache. The pain runs along the course of certain nerves, though it is not confined necessarily to them, but may be somewhat diffuse. Pain is increased or brought on by cold or heat, or pressure on the affected part.

The skin and nerves are sometimes tender or even exquisitely sensitive. Firm pressure, however, is usually not painful. In about half the cases of long standing, *tender points* may be found which correspond to the exit of nerves from a bony canal or the substance of a muscle or fascia. In rare cases there is tenderness over the spine corresponding to the point where the affected nerves arise. Besides feelings of pain, there is often a sense of numbness, cold, tingling, or heaviness of the limb. Vasomotor, secretory, and trophic disturbances may occur; but when these are pronounced one must suspect neuritis or an organic central disease. Muscular spasm is sometimes present; more rarely there is some muscular weakness. The paroxysms of pain may intermit regularly; sometimes they come on every day at the same hour. They are apt to be worse at night. The attacks of the disease often run a course of several weeks or months, and in some forms they last for years.

The painful sensations of neuralgia usually originate in the peripheral sensory neuron. In some cases the posterior spinal ganglion is chiefly at fault (Anstie); in others the irritation affects the entire sensory nerve. The central sensory neurons that carry impulses to the brain are rarely the cause of neuralgia, and local diseases of the cord and brain do not, as a rule, cause pain by irritating these sensory pathways. Still there may be neuralgic pain from this cause; and "central nervous pains" have been observed in brain tumors and after brain hemorrhage or softening.

Pathology.—Many cases that used to be called neuralgia are now known to be forms of neuritis, *e.g.*, sciatica and brachial neuralgia. Other forms are sometimes due to impaired nutrition of the neuron from an obliterating arteritis (tic douloureux); still others are due to the irritation of nerves from the diathetic poison of gout, rheumatism, and diabetes, or to extrinsic poisons, such as alcohol, lead, and arsenic. In these cases the sensory nerves of the nerve sheaths (nervi nervorum) are affected. There remain many cases in which the trouble shifts from one locality to another, or in which no special local or general irritation can be discovered. In these cases we assume that the pathogenic focus is in the spinal or cerebral sensory neurons.

There are some forms of neuralgia which may be called "reminiscent" or "hallucinatory." The patient, who is an impressionable and sensitive person, has had a genuine cause for neuralgic pains; but this, after lasting some time, has ceased, while the painful impressions continue to remain in the cerebral cortex. The

neuralgia is a morbid habit of feeling pain. Such neuralgias are promoted often by the use of morphine.

Diagnosis.—This is based on the fact that neuralgic pains are shifting, paroxysmal, follow the course of nerves, are accompanied often by tender points and not accompanied by signs of organic nerve disease, such as paralysis, anæsthesia, and tenderness over the nerve trunks. Thermic sensations of burning or cold are rarely neuralgic, but are due to neuritis.

Prognosis is good, except for the reminiscent neuralgias, the neuralgias of hysterical and neurasthenic persons, who are thoroughly anæmic, debilitated, aged, and broken down morally and physically. The neuralgias of the degenerative period of life are also very obstinate.

The treatment will be discussed under special heads.

NEUROSES OF THE NERVES OF SPECIAL SENSE.

The Olfactory Nerve.

Anatomy.—The olfactory nerves consist of a number of peripheral fibres that arise from the olfactory bulb, pass through the cribriform plate of the ethmoid bone, and are distributed to the mucous membrane of the superior and middle turbinated bones

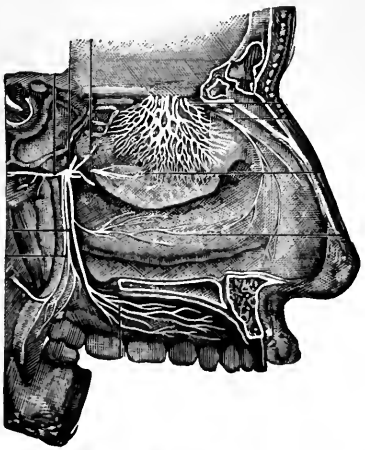


Fig. 83.—Showing the Olfactory Nerve, also a Portion of the Trigeminus.

and the upper part of the nasal septum. The olfactory bulb is with its associated parts really a subdivision of the brain (rhinencephalon) and not a true nerve. For many reasons the anatomy of the rhinencephalon has a peculiar interest. It is developed from a secondary division of the first cerebral vesicle. It includes

in man, from before backward, the bulb and peripheral nerves, the olfactory tracts, and their lateral roots with the gray root or trigonum between them, the hippocampal convolution and cornua ammonis, part of the convolution of the corpus callosum, the nerves of Lancisi, and the anterior commissure. Recent studies of the anatomy of the olfactory bulb and its nerves show that this apparatus is very analogous to that of the retina. Beginning with the nerve cells of the nasal mucous membrane, it is found that these are bipolar, and are analogous to the cells of the posterior spinal

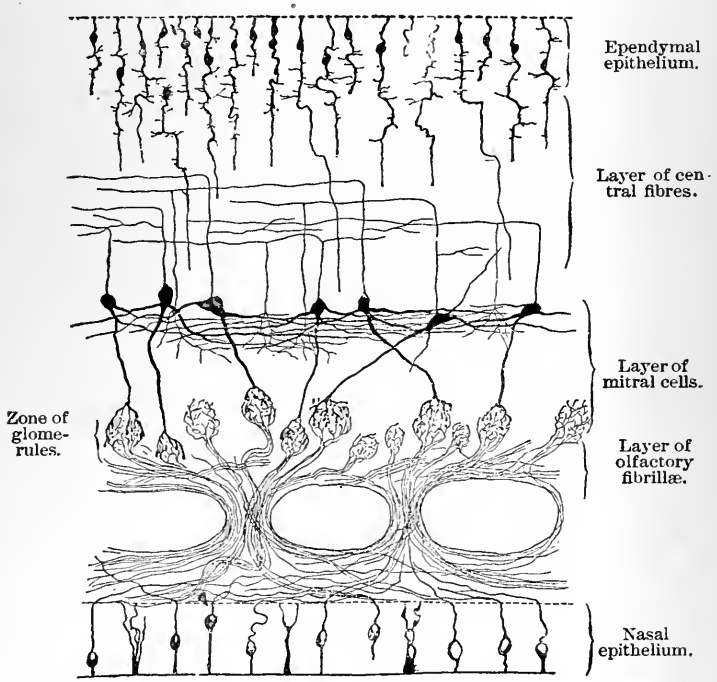


FIG. 84.—PRINCIPAL CONSTITUENT ELEMENTS OF THE OLFACTORY NERVE OF A MAMMAL (Van Gehuchten).

ganglia; they send peripheral processes between the epithelial cells, and these processes receive the olfactory stimulus; the other and central processes pass up through the ethmoid bone to the bulb. These form the so-called nerves of smell. They end in fine expansions which unite with similar terminal expansions from a deeper layer of cells. The tangle of fibres forms what are known as the glomerular bodies. Next is a layer of peculiar cells, called from their shape mitral. They have neuraxons which become part of the olfactory tract. Deeper still is a layer of granular and central fibres, the former being peculiar in having no axis cylinders. These cells re-

semble the spongioblast cells of the retina and are associative in function. The central olfactory fibres, which are now neurons of the third order—that is to say, they are the third from the peripheral olfactory cell—pass to the hippocampus, cornu ammonis, and convolution of the corpus callosum. Through cells in these areas they are put in connection with the optic thalamus and with the motor tracts. The olfactory nerves do not decussate.

The sense of smell is rudimentary in man, yet it is still the sense by which we can appreciate the most attenuated matter; for the trillionth of a grain of mercaptan is able to awaken a sensation in the mind, but has weight and dimensions so infinitely minute as to be quite beyond the power of the imagination to grasp. According to Valentin, we can perceive $\frac{1}{1200000}$ of a grain of oil of roses. According to Fischer and Penzoldt, one can perceive $\frac{1}{27000000000}$ of a grain of mercaptan. I have found that one can perceive the odor from 4 cm. of a solution of oil of cloves, 1 to 100,000. Matter to be perceived as odor must be in a gaseous form. Odorous sensations co-operating with taste sensations form "flavor." Variety in odorous sensations depends probably upon the rapidity of molecular vibrations as in the case of light; and substances having similar relations in vibration have similarity in odor (Haycraft). Males have a more delicate sense of smell than females (Nicolls and Bailey). The keenness of the olfactory sense is lessened in the insane and criminal classes.

Anosmia.—The olfactory nerve is affected clinically by loss of function, or anosmia; increased sensitiveness of function, or hyperosmia, and perversions of function, or parosmia.

Anosmia is far the most common disorder of olfaction.

Etiology.—Its usual cause is disease of the mucous membrane of the nose. Injuries, inflammations, and tumors affecting any part of the course of the nerve, its bulb, or central fibres, may also cause it. Unilateral cortical lesions in the uncinate gyrus may lead to partial loss of smell. It will be not entire, because each nerve receives fibres from both hemispheres. Paralysis of the fifth or seventh nerve may indirectly cause some anosmia. Primary degenerative changes and excessive olfactory stimulation cause anosmia. There may also be a congenital absence of the nerves. Anosmia occurs sometimes as a pure neurosis in hysteria or in neurasthenic states.

Diagnosis.—This is made by test odors. To test the sense of smell, a bottle of oil of cloves or of some familiar non-irritating odor may be used. To detect quantitative disturbance one may use six phials containing oil of cloves, in purity and in watery mixture of 1 to 10, 1 to 100, 1 to 1,000, 1 to 10,000, and 1 to 100,000. Special olfactometers have been devised. The sense of smell for

any single odor is lost in about three minutes, but returns after one minute's rest.

Treatment.—For functional anosmia, snuffs containing strychnine gr. $\frac{1}{30}$ and gum acacia ʒ ij. can be used. Weak galvanic and faradic currents are recommended. Usually there is in anosmia a local catarrhal condition of the nose which requires treatment.

Hyperosmia occurs only rarely and then in neurasthenic, hysterical, or insane persons. In the latter it is more often a psychical phenomenon than a peripheral disorder. Hyperosmia can be cultivated, and this is done sometimes by the blind and by those engaged in certain pursuits, such as tea tasting and wine tasting.

Hallucinations of smell occur in the insane, as just mentioned, and a few cases of epilepsy are reported in which the cause was a stench. When all olfactory sensations are disagreeable the condition is called kakosmia.

Parosmia is a not infrequent condition. In it everything smells alike to the patient, and the odor smelled is perhaps a peculiar or offensive one. This condition may be due to local disease, but is often a symptom of hysteria or neurasthenia.

The Optic Nerve.

Anatomy.—The optic nerve is not a true peripheral nerve but a tract of the brain, and it connects the retinal cells with the brain proper. Like other brain tracts its fibres have a myelin sheath but no neurilemma. The real origin of the nerve is in the retina, just as the olfactory nerve arises in the peripheral cells of the olfactory mucous membrane and the spinal sensory nerves arise in the spinal ganglia.

The retina is a nervous tissue formed essentially of three layers of nerve cells. From without inward they are: the layer of visual cells, the layer of bipolar cells, and the layer of ganglionic cells. This subdivision is shown in the diagram (Fig. 85). These different layers may be subdivided so as to give the following layers from without inward:

1. The layer of rods and cones. } Forming the layer of visual cells.
2. The external granular layer. }
3. The external molecular layer. } Forming the layer of bipolar cells.
4. Internal granular layer. }
5. Internal molecular layer. } Forming the layer
6. Ganglionic layer, with the fibres of the optic nerve. } of ganglion cells

The layer of visual cells is subdivided, as seen in the figure, into that of the rods and cones externally and that of the external granular internally. This is, however, practically a layer made up simply of bipolar nerve cells with prolongations more or less long

which run to the external surface of the retina and there form a series of bodies known as the rods and cones.

In the layer of bipolar cells are layers of cells with processes running horizontally, and in the internal molecular layer are larger horizontal cells, called by Cajal spongioblasts. There are also in the retina terminal arborizations of cells whose origin is in the thalamus, corpora geniculata, or anterior tubercles. These carry impulses *to* the retinal cells (Fig. 85, *s*).

The neuraxons of the ganglionic cells send fibres which unite to form the optic nerve.

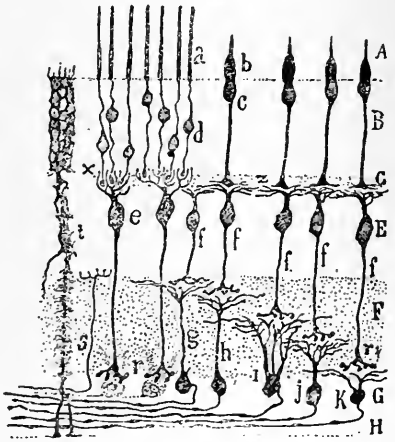


FIG. 85.—TRANSVERSE SECTION OF A MAMMALIAN RETINA. *A*, Layer of rods and cones; *B*, bodies of visual cells (external granular); *C*, external molecular layer; *E*, layer of bipolar cells (internal granular); *F*, internal molecular layer; *G*, layer of ganglionic cells; *H*, layer of optic-nerve fibres; *a*, rod; *b*, cone; *c*, body of the cone cell; *d*, body of the rod cell; *e*, bipolar rod cells; *f*, bipolar cone cells; *g*, *h*, *i*, *j*, *k*, ganglionic cells ramifying in the various strata of the internal molecular zone; *r*, inferior arborization of the bipolar rod cells, connecting with the ganglionic cells; r_1, inferior arborization of the bipolar cone cells; *t*, epithelial or Müller cells; *x*, point of contact between the rods and their bipolar cells; *z*, point of contact between the cones and their bipolar cells; *s*, centrifugal nerve fibre (Cajal).

The optic nerves each contain about 500,000 fibres. They pass to the optic chiasm, where about one-third of the fibres cross, in man. In lower animals the decussation is greater. Those fibres which do not cross come from the outer or temporal third of the retina; those which do cross come from the internal or nasal two-thirds. A few fibres pass from one optic centre in the brain along the posterior border of the optic chiasm to the centre on the opposite side (commissure of Gudden).* After leaving the chiasm, the fibres form the *optic tract*. The tract curves up and back around the crus cerebri, and divides into a lateral and mesial root.

* There is still very high authority for denying the partial crossing of the optic nerve (Michel, Kölliker).

These roots connect with the external geniculate body, the anterior tubercles of the corpora quadrigemina, and the posterior ganglion of the thalamus or the pulvinar. These ganglia are called the primary optic centres. Through the anterior tubercles of the corpora quadrigemina, and by other means, the optic nerve is connected with the oculomotor nerve, and thus reflex movements of the pupils, lids, and eyeballs are brought about.

From these primary optic centres, fibres enter the posterior part of the internal capsule, curve up and back toward the occipital lobes, forming the optic radiations of Gratiolet.

They are finally distributed to the cortex of the occipital lobe, and in man chiefly to the cuneus and the parts about the calcarine fissure.

It will be seen that each retina is connected with the occipital lobe of both hemispheres; further, that the outer or temporal half of each retina is connected with the occipital lobe of the same hemisphere, and the inner or nasal half of each retina with the occipital lobe of the opposite side. The upper part of each retina seems to be connected with the lower part of the cuneus, and *vice versa* (Fig. 86).

Other connections exist by which the optic centres on the two sides are connected with each other and with other cranial nerves in the medulla.

The optic nerve is a nerve of special sense of vision and has no other function except that of an excito-reflex character.

Diseases of the Optic Nerve.

The optic nerve may be affected by nearly all forms of pathological change. For the neurologist, however, the especially important conditions are inflammations, degenerations, injuries, and functional disorders. Inflammation of the nerve, or optic neuritis, may occur as a papillitis or inflammation of the head of the nerve, a neuro-retinitis or descending neuritis, a perineuritis, or a retrobulbar neuritis. Perineuritis is rare. Neuro-retinitis and papillitis are closely associated clinically and pathologically (Noyes), so that practically only two forms of neuritis need be discussed separately.

Papillitis and Neuro-retinitis—*Etiology.*—This condition is seen by neurologists in connection with brain tumors, brain abscess, meningitis, and occasionally multiple neuritis. The other causes are nephritis, diabetes, infectious fevers, lead, and severe hemorrhages. The disease occurs at all ages and in both sexes. In brain tumors it occurs in two-thirds of the cases, and especially often in cerebellar tumors. It occurs in eighty per cent of cases of tuberculous meningitis.

Symptoms.—Subjective symptoms are often not present. The vision may remain good for a long time. In other cases there are concentric limitation of the visual field, loss of color sense, and scotomata. For a description of ophthalmoscopic changes, the reader is referred to special text-books. It is in this condition that "choked disc," which is a papillitis with much serous infiltration, occurs. The changes are less striking in neuro-retinitis. The disease may affect one or both nerves. In brain disease both nerves are usually involved.

Pathology and Pathological Anatomy.—The process is usually

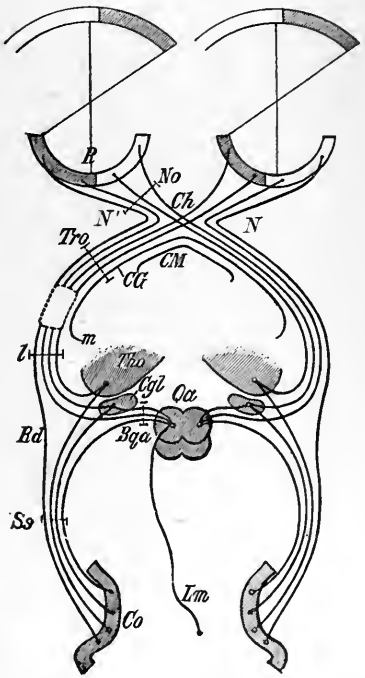


FIG. 86.—THE OPTIC NERVE AND ITS CENTRAL CONNECTIONS (modified from Obersteiner). *R*, Retina, dark on the side connected with left hemisphere; *No*, optic nerve; *Ch*, chiasm; *Tro*, optic tract; *CM*, Meynert's commissure; *CG*, Gudden's commissure; *Tho*, thalamus; *Cgl*, corpus genic. ext.; *Qa*, corpor. quad.; *Ss*, optic radiations; *Co*, occipital cortex; *Lm*, mesial lemniscus. (The shading of the retina and visual field should just reach the vertical lines.)

subacute or chronic. Congestion, exudation, small hemorrhages, and collections of leucocytes occur. The sheath of the nerve just back of the globe is often distended with a serous exudate. After a time the nerve fibres atrophy, connective tissue proliferates and takes their place, and we have a secondary optic atrophy.

The process is essentially peripheral, but it extends back with lessening intensity into the trunk of the nerve. The purely mechanical theory of neuritis, that it is due to compression, cannot be accepted in the light of modern pathology. It is probable that the neuritis results from an irritating serous fluid which extends down the sheath of the nerve, this sheath being a prolongation of the arachnoid cavity. Mechanical causes lead to constriction, accumulation of the fluid, and compression of the nerve at its periphery, and hence to inflammation. Sometimes, at least, the irritating fluid contains microbes or microbic poisons.

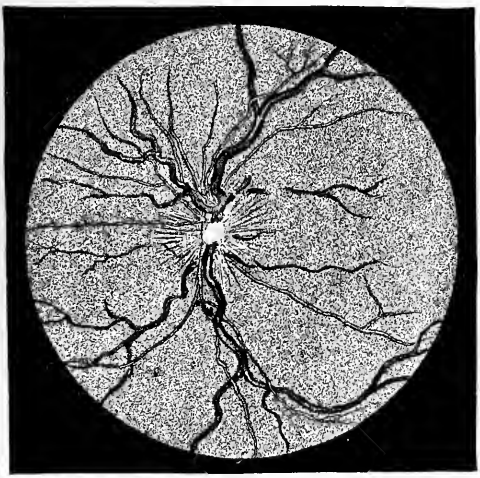


FIG. 87.—NEURO-RETINITIS (Jaeger).

RETROBULBAR NEURITIS.—In this disease the lesion lies chiefly behind the globe. Its causes are especially toxæmia from alcohol and tobacco. It is also due to rheumatic influences, syphilis, lead, and diabetes.

In the acute cases there is usually rather rapid loss of sight, with some pain and tenderness. The ophthalmoscopic changes are relatively slight. In chronic cases, which are usually toxic in origin and due to alcohol or tobacco, or oftenest to both, there are color scotomata or absolute scotomata and amblyopia. There is no pain. The condition is known as tobacco or alcoholic amblyopia.

The *prognosis* of neuritis varies with the cause. If this is removable, as in the toxæmias, recovery is the rule. This is a proof that in neuritis the connective tissue is the part chiefly involved,

for a destroyed or atrophied optic nerve does not recover. In many cases, however, atrophy follows the neuritis.

The *treatment* is based on the cause. In acute cases one may use cups, salicylates, the iodides, and mercury; later, the iodides and strychnine. Perfect rest to the eyes should be enjoined.

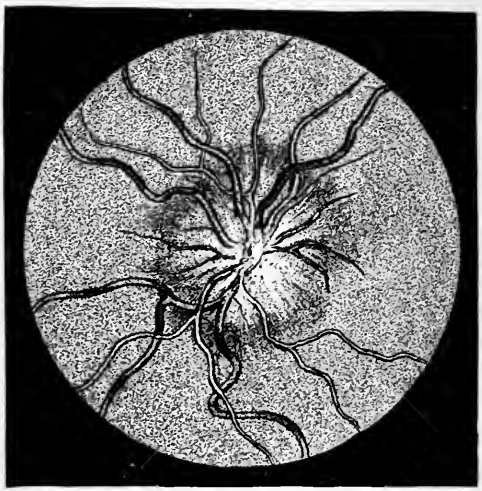


FIG. 88.—PAPILLITIS, "CHOKED DISC," IN A CASE OF CEREBRAL TUMOR.

DEGENERATION OF THE OPTIC NERVE, OR OPTIC ATROPHY.—This condition may be primary or secondary. Secondary atrophy is usually the result of a neuritis. I shall describe here *primary optic atrophy.*

Etiology.—It occurs oftener in men than in woman (three to one). It occurs as part of locomotor ataxia in less than half the cases; other degenerative diseases of the cord, like multiple sclerosis, may accompany it. Next to the spinal cord, it occurs oftenest with degenerative diseases of the brain, such as multiple sclerosis and general paresis. Hemorrhages, alcoholism, and lead may be causes. It may occur without known cause.

The *symptoms* are those of gradual decrease of acuity of vision, concentric limitation of the visual field, loss of color sense, dilatation and immobility of the pupil. The sense of sight may remain good for a long time. Ophthalmoscopically, the nerve disc is opaque, grayish, or dirty looking, and often has a cup-shaped or "cupped-disc" appearance. The vessels are smaller and few in number.

The *pathology* and *pathological anatomy* are that of a parenchymatous degeneration with loss of nerve fibres, which are replaced by connective tissue.

The *prognosis* is almost uniformly bad.

The *treatment* is that usually of the cerebral or spinal disease. Mercury, iodides, strychnine, phosphorus, and iron are given. Warm baths and salicylate of soda sometimes have a temporarily good effect. Strychnine in physiological doses gives, however, the best symptomatic results. Electricity is not of any use. Stretching the nerve does no good. Eserine, pilocarpine, and santonin, or nitrate of silver may be tried.

The optic nerves and their primary and cortical centres are subject to various other diseases. So far as these are organic, they will be described in detail under the head of brain diseases. But there are certain symptoms often of functional origin which are best described here. These are: (1) Amblyopia and amaurosis; (2) retinal hyperæsthesia and dysæsthesia; (3) hemianopsia.

Amblyopia and Amaurosis.—Amblyopia is a partial loss or dimness of vision, there being no observable lesion of the eye, or its nerves. Amaurosis is a total loss of vision, also without observable cause.

Etiology.—The causes are injuries and shocks, hysteria, migraine, concussion of the brain, lightning stroke, and severe hemorrhages. There are also certain toxic causes, chiefly alcohol and tobacco, quinine, and salicylic acid. Other causes are glycosuria, uræmia, and reflex irritations, especially of the trigeminal nerve. Night blindness and snow blindness are forms of functional amblyopia.

The *symptoms* are diminution or loss of vision, usually sudden, temporary, and involving both eyes. Amblyopia in hysteria is usually greater in one eye and associated with concentric limitation of the visual field and disturbance of color sense.

Underlying amblyopia there may be minute hemorrhages in the brain, causing temporary pressure, or a vascular spasm, causing anæmia.

The *prognosis* is usually good.

The *treatment* is purely a causal one. In most cases one must examine for drug poison, uræmia, diabetes, migraine, or a hemorrhage.

Retinal or ocular hyperæsthesia is a condition in which the eye is abnormally sensitive to light. It may be due to exposure to extreme light or to seclusion in a dark room. The neurologist sees it oftenest as a symptom of hysteria (*vide* Hysteria) and neurasthenia and perhaps in hypnotic states. It occurs in my-

driasis and albinism. It is not to be confounded with photophobia due to irritation of the conjunctiva.

Nyctalopia, or the condition of seeing better in a dim light, is a form of the disease.

Hemianopsia, or half-sightedness, or hemianopia, a condition in which there is a blindness of one-half the visual field, may be due to a functional or organic disorder of the nerve or its centres. It is a symptom of many lesions and conditions, and can be described only generally here.

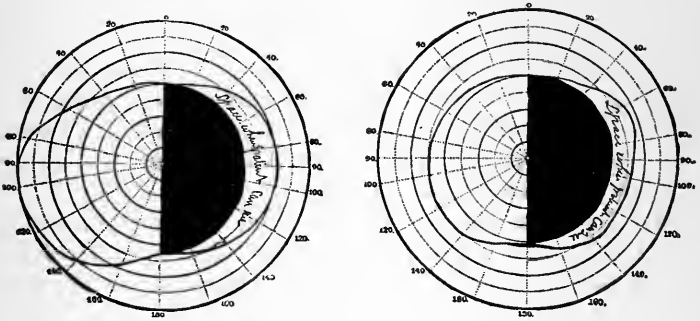


Fig. 89.—Showing Lateral Homonymous Hemianopsia. This is not quite complete, there being a space on the blind side where the patient can see (Noyes).

Etiology.—Its principal functional causes are migraine, lithæmia, gout. Its organic causes are tumors, inflammations, softenings, or hemorrhages involving part of the optic nerve or its central connections.

Symptoms.—Various descriptive terms are used to indicate the character of the hemianopsia. In lateral hemianopsia a vertical half of the field is involved. In lateral homonymous hemianopsia there is half-blindness on the left or right side of each eye, as the case may be (Fig. 89). In temporal hemianopsia the outer halves of the eyes, and in nasal the inner halves, are involved. The upper or lower segments, or irregular segments of the visual field may be involved.

These various forms of hemianopsia depend upon the location of the lesion which cuts into and destroys the optic fibres in their course from the eye to the visual centre in the occipital cortex. The mechanism will be understood when it is remembered that each occipital lobe is supplied by nerve fibres from one-half of the retina of each eye. A cut shows this better than any description (Fig. 86). In temporal hemianopsia the lesion must be at *Ch*, in front of the chiasm. In bilateral nasal hemianopsia it must be double

and at N and N'. In lateral hemianopsia the lesion must lie farther back than the chiasm, in the tract, the primary centres, the optic radiations, or occipital lobes.

In hemianopsia from disease of the nerve as far back as and including the primary centres in the optic thalamus and corpora quadrigemina there is a loss of light reflex when a ray of light is thrown upon the blind side of the retina, but the pupil still contracts when light is thrown on the sensitive side of the retina. This phenomenon is called "Wernicke's hemiopic pupillary reaction." If in hemianopsia the light reflex is preserved, the lesion is back of the primary centres and involves the optic radiations or cortex.

A test for the condition of hemianopsia in its early stage, and one that is useful in stupid or partially comatose patients, is the following: When the finger is suddenly brought in front of the eye on the sound side, there is a wink; if brought in front from the blind side, the orbicularis does not contract.

Hemianopsia is almost always the sign of organic disease. It is not found in hysteria, but does occur in migraine and lithæmia. It is best made out and recorded by means of the perimeter.

Its course and treatment depend upon the cause.

Sensory Neuroses of the Trigeminal Nerve.

Anatomy.—The trigeminus or fifth nerve is one of the most extensively distributed and most delicately sensitive nerves of the body. Its sensory branches represent the atrophied and lost sensory roots of the third, fourth, sixth, seventh, and twelfth cranial nerves (Gaskill). The trigeminal nerve is a mixed nerve. It has two nuclei of origin: a central nucleus for the motor part, and a peripheral nucleus for the sensory part. The motor nucleus has two parts: a chief nucleus lying deeply in the substance of the pons Varolii, and an accessory nucleus, which consists of a long tract of gray matter, known as the descending root and lying in the upper part of the dorsal portion of the pons. It passes down along the side of the aqueduct of Sylvius. There has been much discussion as to the exact function of this root or nucleus, but the most recent studies have shown quite conclusively that it is motor in function (Kölliker, Cajal). The sensory root of the trigeminal has its origin in the Gasserian ganglion, which is composed of unipolar cells, like those of the spinal ganglia. The axis-cylinder processes of these cells bifurcate; the external branches pass outward and become part of the peripheral sensory root; the internal branches pass into the substance of the pons, and there give off ascending and descending branches. The ascending branches are short, and pass to a terminal nucleus, known heretofore as the sensory nucleus

of the trigeminal. The descending branches pass down to the pons and medulla, as far as the cervical part of the spinal cord. In their course they give off terminal filaments, which come in relation with sensory nerve cells, and the whole forms a column of matter, known as the *ascending root* of the trigeminus.

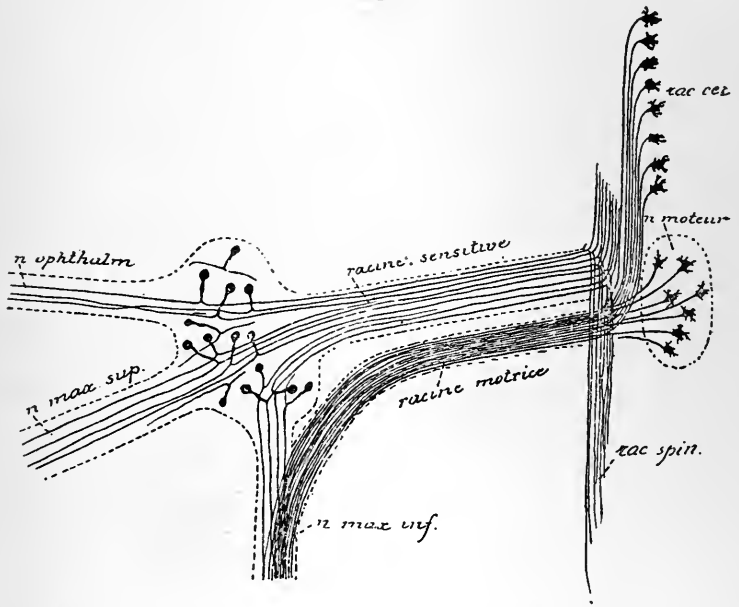


FIG. 90.—SHOWING THE MODE OF ORIGIN OF THE TRIGEMINAL NERVE, AND THE RELATIONS OF THE MOTOR AND SENSORY ROOTS. *rac. cer.* is the cerebral or motor root and its fibres go to the inferior maxillary branch; *rac. spin.* is the spinal or ascending root. The cut shows the Gasserian ganglion giving origin to the sensory fibres (Van Gehuchten).

The nuclei of the trigeminus reach the whole length of the pons and medulla, and are coextensive with the origin of all the other cranial nerves (Fig. 91). Hence the frequency with which its disorders are complicated with those of these nerves. Its cortical origin is probably in the lower part of the pre- and post-central convolutions.

The trigeminus supplies sensation to the face, conjunctivæ, nose, the frontal and maxillary sinuses, the teeth, the palate, tongue, and part of the upper pharynx; also to the scalp as far back as the vertex, and to the external auditory meatus (Fig. 93). The distribution is not always the same and is helped by fibres from the cervical nerves. Entire removal of the Gasserian ganglion in man may give, for example, anæsthesia only over the areas shown in Fig. 92, in which anæsthesia is not complete in areas *f* or *a*. It gives sensation also to the anterior three-fourths of the dura mater, the falx, and probably the tentorium. The pia and arachnoid are not sensitive.

The posterior fossa and the occipital part of the dura mater are supplied by the vagus. The trigeminus also supplies the above-named parts with trophic, vasomotor, and secretory fibres. The vasomotor fibres are brought to it, in part, from the medulla and cervical spinal

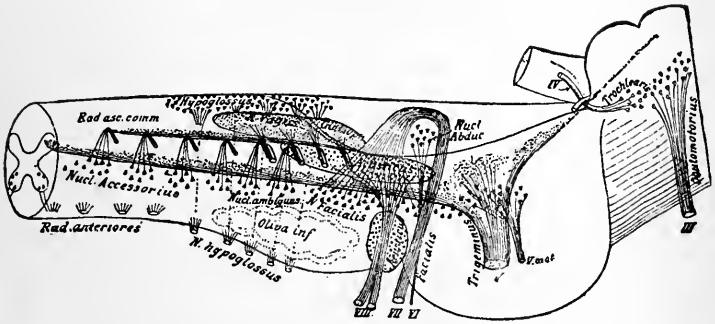


FIG. 91.—SHOWING THE ORIGIN OF THE CRANIAL NERVES AND THE EXTENT OF THE ORIGIN OF THE TRIGEMINUS (Edinger).

cord *via* the sympathetic; the secretory fibres have the same origin. An exception is to be made of the lacrymal secretory fibres which are brought by the motor nerves of the eyeball. The opinion, based largely on physiological experiment, that the trigeminus sends

FIG. 92.—SHOWING AREA OF ANÆSTHESIA AFTER REMOVAL OF GASSERIAN GANGLION (Krause).

trophic fibres to the conjunctivæ and cornea, is denied by Krause, who reports many successful cases of entire extirpation of the Gasserian ganglion without any inflammation of the eye following.

The trigeminus supplies motion to the muscles of mastication, viz., the two pterygoids, the temporal, masseter, mylo-hyoid, and anterior belly of the digastric. The sensory neuroses of this nerve are neuralgia, paræsthesia, and anæsthesia.

NEURALGIAS OF THE TRIGEMINUS.—The trigeminal nerve is subject to two types of neuralgia, viz.:

1. The symptomatic form.
2. Tic douloureux.

1. The *symptomatic neuralgias* are by far the most frequent.

They are called supra-orbital, infra-orbital or supramaxillary, inframaxillary or dental, and mixed forms. The most common type is the supra-orbital; next, the mixed form.

Etiology.—The female sex is oftenest affected; most cases are seen in the first half of life; most attacks occur in the winter and spring. The left side is oftener affected. The second and third branches of the fifth nerve are most susceptible to rheumatic influ-

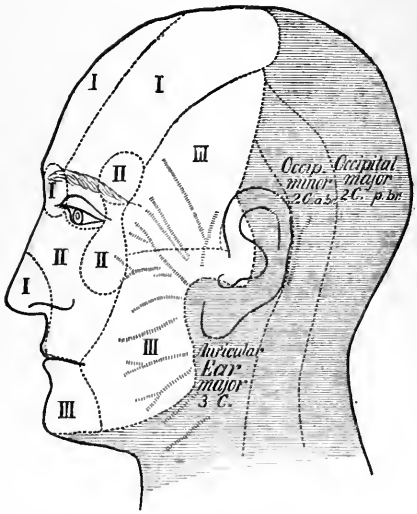


FIG. 93.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE FACE. *I, II, III*, First, second, and third branches of the fifth. The shaded part is supplied by the cervical nerves; 2 *C.a.br.*, second cervical anterior branch; 2 *C.p.br.*, second cervical posterior branch.

ences, the first branch to malarial and septic poisons. Dental disorders naturally are frequent factors in neuralgia of the second and third branches. Anæmia, exposure, child-bearing, and other depressing influences are factors in causing these neuralgias. Ocular and nasal disease may cause pain in the supra-orbital nerve. Gout, diabetes, syphilis, malaria, hysteria, epilepsy, trauma, rheumatism, all may be factors in causing trigeminal neuralgias.

Symptoms.—The pains of trigeminal neuralgia are sharp and intense, with exacerbations and remissions. The pain often lasts for days without entirely ceasing. It then goes away and may not return

for a long time. The characteristic of neuralgia is that it stays till its cause is removed. In supra-orbital neuralgia there is sometimes great œdema of the lids and the parts about, together with suffusion of the eyes. There are tender points over the course of the nerves. The tender points in supra-orbital neuralgia are supra-orbital, palpebral, and nasal; in infra-orbital neuralgia, nasal, malar, and gingival; in infra-maxillary neuralgia, inferior dental and temporal. In the mixed forms we find various combinations of the above. There is often also a tender point over the parietal eminence and vertex.

The pain may radiate to the ear or occiput, or it may be located in the orbit. Dental irritation may also cause an otalgia or a neuralgia in the upper branches of the fifth.

There is sometimes a dilatation of the pupil, and in severe cases a reflex facial spasm occurs.

Tic Douloureux (*Prosopalgia, Fothergill's Neuralgia, Epileptiform Neuralgia*).—Tic douloureux is a special form of trigeminal neuralgia occurring in middle or advanced life, unusually severe in its symptoms and obstinate in its course. It ought to be distinguished sharply from the ordinary forms of trigeminal neuralgia. These latter are symptomatic pains almost altogether; while tic douloureux is a special disorder, dependent upon changes in the nerve itself.

Etiology.—It occurs, as a rule, in persons who are over forty, and is seen in the very aged. It is, indeed, almost the only neuralgia which old people have. It is brought on by exposure, overwork, and depressing influences; sometimes, perhaps, by local diseases of the teeth and jaws. It occurs in men and women in about equal frequency, in my experience. Other observers find it oftener in women.

Symptoms.—It is characterized by intense darting pains, which usually start in the upper lip and by the side of the nose. From here they radiate through the teeth or into the eye and over the temple, brow, and head. They are confined to one side of the head. During a paroxysm the face usually flushes, the eyes water, the nose runs, and the patient assumes an expression of the greatest agony. The attack lasts for a few minutes, then becomes somewhat less, but the pain rarely ceases entirely. A breath of cold air, speaking, eating, putting out the tongue—all bring on paroxysms. The pains are worse in winter and often become less or cease during summer. Occasionally they come on for a few months every year, usually during the spring. The pains are always limited to one side of the face and are centred chiefly in one branch of the nerve.

oftenest in the supramaxillary and next in the inframaxillary. They may spread so as to involve the whole of one side of the face and tongue.

Spasmodic movements of the face, tongue, or jaws may be associated with the pain.

Examination rarely reveals any objective trouble, but in a few cases some anæsthesia may be noted.

Pathology.—The disease is usually a degenerative one, and probably is due to irritative and atrophic processes occurring in the nerve and its ganglion. A low grade of neuritis, perhaps from alveolar disease, has been found sometimes, but as a rule the nerve does not appear much changed. The arteries supplying the nerve, however, often undergo the changes of endarteritis, their calibre is much lessened, and the nerve cannot get its proper supply of blood. Thus an obliterative arteritis underlies some cases of the disease.

Treatment.—In cases which occur in old people, the use of nitroglycerin given in doses of gr. $\frac{1}{100}$ q. 2 h. sometimes has a happy effect. A very good remedy is crystalline aconitia given in doses of gr. $\frac{1}{200}$ until its physiological effect is obtained. Besides these measures, galvanism daily, iodide of potassium in large doses, gelsemium, croton chloral, codeine, external applications of menthol, freezing with chloride of methyl, and heat—all may be found useful. The common practice of pulling out all the teeth is almost always unsuccessful, and ought not to be undertaken without specially good reason. Tonics containing iron, phosphorus, quinine, or arsenic are generally helpful, and should always be given after a course of specifics. In younger patients the remedies recommended under the head of Migraine and Headache may be given. Change to a warm, equable climate may be tried; it is not a certain resource. I have found that in cases not of over four or five years' duration, rest in bed with massive doses of strychnine sometimes effects striking cures. The drug should be given hypodermically in doses of gr. $\frac{1}{30}$ once daily, gradually increased until gr. $\frac{1}{6}$ or $\frac{1}{5}$ is reached. This is repeated four days and then the amount gradually reduced. The patient must be kept rigidly quiet and the full course persisted in. After a month iodide of potassium and iron are given. The treatment may have to be repeated with lessened rigor. The treatment by large doses of opium, gr. iii. to vi. daily, is uncertain and often dangerous.

Finally, surgical interference may be necessary. The removal of the nerve at as deep a point as possible is the only operation to be seriously entertained. This sometimes causes cure, but, as a rule, the pain comes back in six or twelve months. Even such a

respite, however, is often gladly seized upon. Removal of the Gasserian ganglion has been attempted with success. Ligature of the common carotid has been tried also, but of late years the operation has been generally abandond. Simple drilling out of the infra-orbital nerve with a dental probe made of piano wire has given long relief.

There are numerous methods of operating upon the different branches of the trigeminus. For buccal neuralgia, Zuckerkandl has devised a method. For superior maxillary neuralgia, the method of Carnochan, modified by Abbe, is, in my opinion, the best. Others favor Langenbeck's method. Ullmann, Mikulicz, Obalinski, and many others have devised special methods.

Hartley, of New York, has devised an operation by which he enters the middle fossa through an opening in the temporal bone, thus reaching the root of the nerve. Rose, Krause, and others have reported many successes following this rather serious operation.

Trigeminal Paræsthesia.—Sometimes persons suffer from peculiar numbness, thrilling, or formication in the course of the trigeminus. The sensation may be nearly constant and excessively annoying. It never amounts to actual pain.

It occurs in anæmic, nervous, and hysterical persons. It is to be regarded as an abortive form of neuralgia, and so treated.

Trigeminal Anæsthesia.—This occurs from various pathological lesions in the course of the nerve or in its nuclei. The most common organic cause is syphilitic disease of the membranes at the base of the brain. Trigeminal anæsthesia occurs together with anæsthesia of other areas in hysteria and in organic disease of the nerve centres. It is sometimes noted in tic douloureux and facial hemiatrophy.

Herpes, flushing, pallor, lacrymation, salivation, are all symptoms of disturbance of the trophic, vasomotor, and secretory fibres running in the trigeminal nerve. They are usually, if pathological, only concomitant symptoms of other diseases.

Headache (Cephalalgia).

Headache is the name given to attacks of diffuse pain affecting different parts of the head and not confined to the tract of a particular nerve. It usually comes on in paroxysms at various intervals, but may be continuous.

Etiology.—Headache is the most common of nervous symptoms. Ten to fifteen per cent of school children, twenty-five per cent of men, and over fifty per cent of women are subject to it more or less,

though this proportion would be much reduced if migraine were excluded from statistics.

The headache ages are from ten to twenty-five and thirty-five to forty-five; most cases occur between the ages of eight and twenty-five, especially in females. The number of headaches increases gradually from the period five to ten years up to the period fifteen

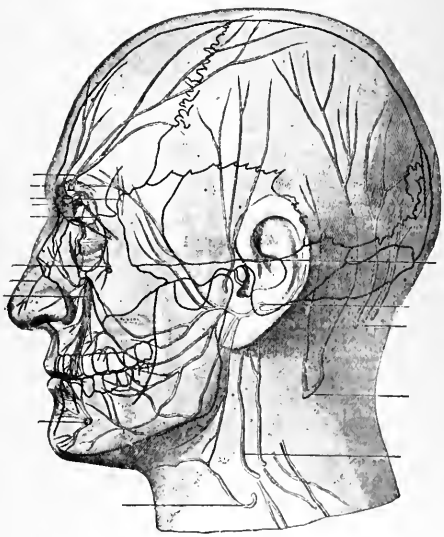


FIG. 94.—SHOWING THE MOTOR AND SENSORY NERVES OF THE FACE (Merkel).

to twenty, then falls till the thirty-fifth year, and rises again till about the age of forty. Early childhood and declining age are practically exempt from chronic functional headaches. Women suffer from it more than men in the proportion of about three to one. It is more frequent in city populations and among the wealthier classes. Headaches are more common in the spring and fall and in temperate climates. Headaches may be classed, in accordance with their causes, as follows:

1. Hæmic or autotoxic causes, in which impoverished or disordered blood is brought to the brain, as in (*a*) anæmia and congestion; (*b*) diathetic states: gout, rheumatism, diabetes, uræmia; (*c*) infections: malaria, fevers.

2. Toxic causes: lead, alcohol, tobacco, etc.

3. Neuropathic states: epilepsy, neurasthenia, hysteria, neuritis.

4. Reflex causes: ocular, nasopharyngeal, auditory, dyspeptic, sexual.

5. Organic disease, including arterio-sclerosis, syphilis, tumors, meningitis, and diseases of the cranial bones. Very frequently several causes act together. The anæmic, dyspeptic, ocular, and neurasthenic are the common forms of chronic and recurrent headache.

Edinger thinks that a large percentage of chronic headaches are due to the presence of certain nodules situated near the origin of the muscles of the back of the head. The location of these is indicated in Fig. 95. Massage by removing these relieves the head-

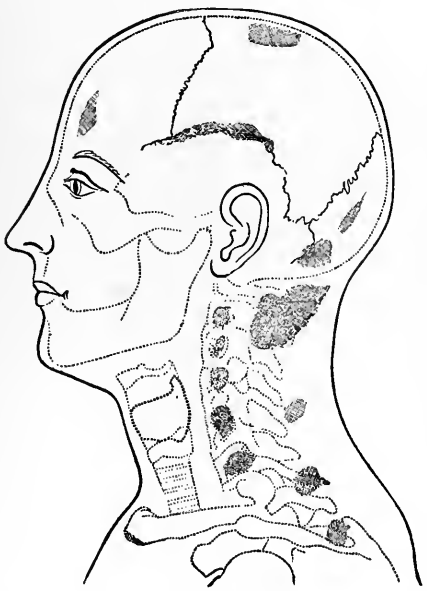


FIG. 95.—LOCATION OF HEADACHE NODULES ACCORDING TO EDINGER.

ache. I have not found these often, and doubt if they have so much importance, but their existence and possible influence should be borne in mind, especially in gouty subjects.

Symptomatology.—Headaches may be classed in accordance with their location and the character of the pain. We have accordingly: 1, frontal headaches; 2, occipital headaches; 3, parietal and temporal headaches; 4, vertical headaches; 5, diffuse headaches and various combinations of the above.

The most common form of headache is the frontal, next the fronto-occipital or diffuse, next the vertical, and then the occipital.

The kind of pain differs with different persons and with different causes. We have: 1, pulsating, throbbing headache; 2, dull,

heavy headache; 3, constrictive, squeezing, pressing headache; 4, hot, burning, sore sensations; 5, sharp, boring pains.

The first form characterizes headaches with vasomotor disturbances, and usually indicates migraine.

The second is usually of a toxic or dyspeptic type. The third is found in the neurotic and neurasthenic. The fourth in rheumatic and anæmic cases. The fifth in hysterical, neurotic, and epileptic cases.

The accompanying diagram shows some of the relations of localized pain to the cause. A large experience both in my own practice and in that of others shows it to be approximately correct.

Headaches may continue for a day or may last for weeks or months. Some persons have headaches only when constipated or

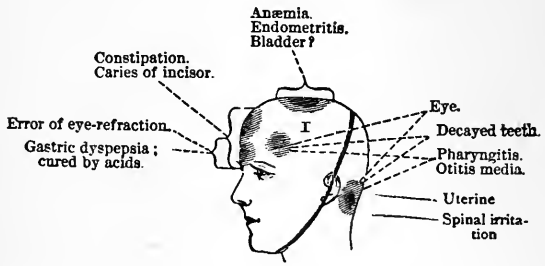


Fig. 96.—Showing the Location of the Pain in Headaches from Different Causes.

bilious, or when they have an attack of indigestion. Others suffer from a little pain nearly all the time, exacerbations occurring at various periods. Neurasthenic and ocular headaches are generally of this type. When headaches are persistent, examination should be made of the eyes, of the nose and sinuses; the patient should be questioned as to syphilis, the continuous use of tobacco, and chronic dyspepsia. The possibility of brain tumor, of pachymeningitis from blows, or sunstroke or chronic alcoholism should be considered.

The persistent headaches not relieved by ordinary treatment are due to eye trouble, anæmia, neurasthenia or spinal irritation, rheumatic nodules, syphilis, or pachymeningitis. Eye strain may cause true migraine or ordinary headache. Eye-strain headache is usually associated with some weakness of eyesight and pains and discomfort about the globe, besides severer pains at times in the brow or occiput. The cause of the eye strain is usually astigmatism and hypermetropia. Occasionally it is due to weakness or lack of balance of the eye muscles.

Headaches may occur regularly every morning on awaking. They are called *morning headaches*, and are a symptom of neurasthenia and lithæmia. They occur oftenest in middle life.

Symptoms Associated with Headache.—The symptoms oftenest associated with chronic and recurrent headaches are vertigo, somnolence, sensations of heat and pressure (cerebral paræsthesias), and nausea. Vertigo goes oftenest with headaches of dyspeptic origin; some of the so-called bilious headaches of early life develop later into attacks of vertigo; this symptom often occurs with frontal headaches. Somnolence occurs oftenest with anæmic and malarial headaches; it may develop also with syphilitic head pains. Nausea I have found oftenest with occipital headaches.

Pathology.—Headaches are to be distinguished from neuralgias and from a special and common form of head pain known as migraine.

Headaches are diffused pains caused, as a rule, by irritations located in or referred to the peripheral ends of the fifth nerve. Their seat is usually within the skull.

Neuralgias, on the other hand, are caused by irritations of the ganglia or trunks of these nerves. The pains are local and confined to the single branches of the nerve.

Migraine is a periodical neurosis in which there is a discharge of nerve force, not only affecting the trigeminus, but often other cranial nerves as well as sympathetic fibres. It is a general disease of which the headache is only one symptom.

The nerves of the dura mater are those most involved in headache. Headaches, when occipital, involve the sensory fibres of the vagus and the upper four cervical nerves. There is no anatomical change in the nerves except in organic headaches. But in many cases the membranes of the brain and their sensory nerves are congested or anæmic.

Diagnosis.—No symptom requires more careful investigation as to its cause than that of headache. The diagnosis is always to be made, not of this symptom, but of its cause. Most of the foregoing description accordingly refers to etiology. It may be quite positively affirmed that headaches which persist for months, are worse in the day, and leave the patient able to sleep at night, to recur on waking, are exhaustion pains and are due to a neurasthenic state. Chronic headaches, worse at night, are usually of specific or organic origin.

It is important, however, to decide whether the case is one of migraine, or neuralgia, or headache. Headache is usually diffuse and bilateral. It is more or less persistent. Migraine comes on paroxysmally, lasts a short time, and then leaves the patient feeling perfectly well or even better than ever. Migraine is often accompanied with nausea, flashes of light, strong pulsations of the head,

vertigo, pallor, or, more rarely, congestion of the face. Neuralgic pains are sharp and shooting; they run along the tract of the nerve, and often are associated with suffusion of the eye and œdema. Tender points are felt.

Treatment.—The constitutional treatment is based upon the etiology. Regulation of diet and digestion, securing a regular movement of the bowels, attention to ocular troubles, abstention from tobacco and alcohol and overwork are the important points requiring attention. Rest is the important point to attend to, as in almost all painful neuroses.

The symptomatic treatment consists in giving antipyrin, antifebrin, phenacetin, salicylate of sodium, caffeine, muriate of ammonia, and sometimes morphine or codeine, and bromide of ammonium.

Antipyrin can be given in doses of gr. v. every twenty minutes till three or four doses are taken. Phenacetin often needs to be given in large doses of ten or even twenty grains. Antifebrin is less trustworthy and must be given in small doses. Exalgin is not a very good or safe remedy. It may be tried in doses of gr. iij. to gr. v. Muriate of ammonia is an excellent remedy given in very large doses, ʒ ss. to ʒ i., well diluted. Menthol in doses of gr. v. to gr. x. in hot water sometimes stops headaches. In headaches from anæmia, caffeine and ammonium muriate are best. In headache from nervous exhaustion, similar stimulating anodynes are usually most efficacious. Combinations of caffeine citrate and salicylate of sodium or benzoate of sodium are often better than the single drug. Caffeine in any case must be given in larger doses than is ordinarily done, *i.e.*, gr. iv. or v. Local applications of a twenty-per-cent solution of menthol, the ice bag, cloths wrung out in hot water, or a piece of sheet lint soaked in chloroform liniment two parts and tincture of aconite one part are efficacious measures. A cathartic, rest in a darkened room, light diet—all these are measures which many patients themselves learn to adopt.

Finally, in headaches from organic disease we have often to resort to iodide of potassium, mercury, and the use of some preparation of opium.

As will be seen, each case of headache requires special treatment and a certain amount of experimentation in order to learn the idiosyncrasy of the patient.

Migraine, Sick-Headache, Hemicrania.—Migraine is a constitutional neurosis characterized by periodical attacks of pain chiefly in the course of the fifth nerve. The pain is often associated with nausea or vomiting, mental depression, vasomotor disturbances such as flushing or pallor of the face, by flashes of light, vertigo, tinnitus

aurium, and in rare cases by partial paralysis of one oculomotor nerve.

It will thus be seen that migraine is more than ordinary headache and unlike an ordinary neuralgia.

Etiology.—The disease is very common in civilized countries and is frequent in America. It occurs oftenest in women in the proportion of about three to one, and it begins in most cases at or a little before the age of puberty. It may begin as early as the fifth or even the second year. It occurs in neurotic families, and there is very often a history of direct inheritance. Other neuralgic troubles, epilepsy, and gout may be found in the family history. The attacks occur oftenest in the winter in our climate. The cases that begin in childhood and early life are sometimes started by overwork at school, but usually no especial cause can be found. When they begin after maturity, a history of excesses in work, injury, shock, or exhausting disease is found. Migrainous patients usually have some refractive disorder of the eye or a weakness of eye muscles, and this condition is one factor in bringing on or keeping up the headaches. Autotoxæmia from uric acid and poisons developed in the intestinal tract is considered an important factor in migraine by some.

Symptoms.—The patient for several days may feel a sense of malaise and depression; usually, however, the prodromal stage lasts only a few hours or a day. The attack often comes on in the morning and gradually increases in intensity until the victim has to give up work and lie down. Sometimes the pain comes on with almost epileptic suddenness and violence, waking a person from sleep or compelling him at once to lie down. *Fulgurating migraine* is the term applied to this type. The pain starts in one side of the head, usually in the forehead, but often in the occiput. It increases and finally may involve the whole head. The pain is of a tense, throbbing, blinding character, increased by jars, light, and noises. It is accompanied by dimness of vision, often by flashes of light or dark or light spots variously colored floating before the eyes. Restriction of the visual field, sometimes in the form of hemianopsia, may occur. Vertigo, tinnitus aurium, confusion of ideas, feeling of stupor, disturbances of memory, are not uncommon; nausea and even vomiting are the rule. The vomited matter is at first chiefly mucus, but it may later become yellow and bitter from the presence of bile. Hence the term "bilious headache," which is an improper one, because the bile is only the result of retrostaltic action from the vomiting. Migraine is not the result of gastric or liver disorder.

The patient's face usually is pale and gives the evidence of acute

suffering. The flushed face is very rare; the distinction between angiospastic or pallid migraine and angioparalytic or congestive migraine is not of clinical value. The pulse is small and hard, and may be lessened in rapidity. The temperature in children often rises.

The attack lasts from six to twelve or twenty-four hours, occasionally even two or three days. As the intensity of the pain lessens

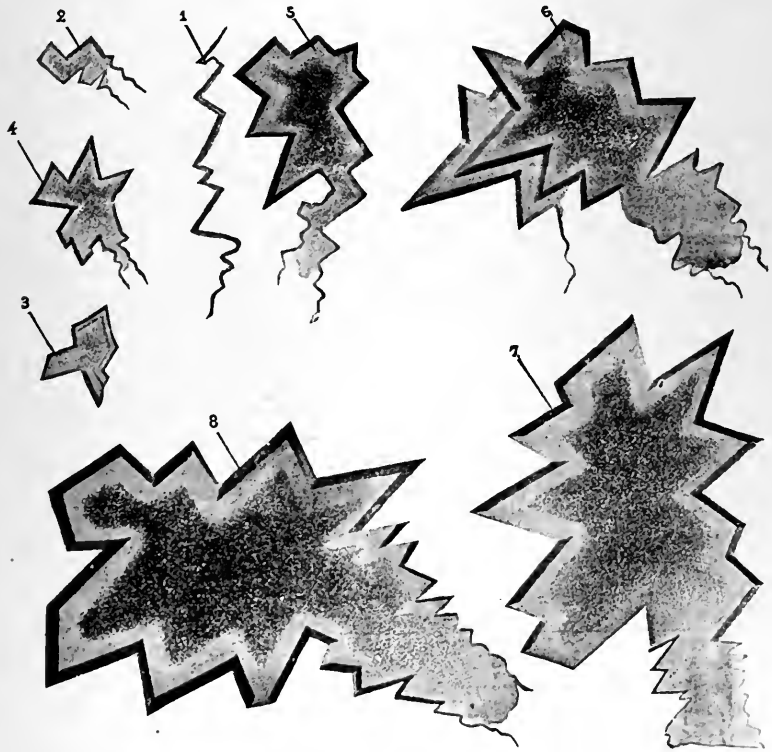


FIG. 97.—SCINTILLATING AND ZIG-ZAG LIGHTS SEEN IN MIGRAINOUS ATTACKS (Babinski).

the patient sinks to sleep, and awakens next morning feeling refreshed and better than before the attack.

The attacks occur at varying periods, fortnightly or monthly, and even weekly. In women they often occur during menstruation. Some women are entirely free from them during pregnancy. At about the time of the menopause in women, and at about the same time of life in men, the disease lessens in severity and as a rule disappears. Some form of neuralgia or some neurosis in rare cases takes its place.

Complicating Symptoms.—Partial oculomotor paralysis, temporary aphasia, slight hemiplegia, heminumbness, peculiar odors or tastes, convulsive movements of the body almost resembling epileptic attacks, occur.

Cases presenting these symptoms are rare. When they occur in one case, however, they always occur in each attack unless it is modified by treatment.

Vicarious Attacks.—Migraine is sometimes associated with epilepsy, locomotor ataxia, or insanity; that is to say, persons in early life have had migraine and later developed the diseases mentioned. The relation between these diseases, however, is not so close as some writers would lead us to infer. The attack of migraine can be sometimes replaced by an attack of gout, or visual disorders, or other sensory symptoms, or even by an oculomotor paralysis. Sometimes, instead of a fully developed attack, the patient has a sense of mental depression, with confusion of ideas. Cases have been reported in which acute mania took the place of the headache.

Types.—Writers have described the angioparalytic and angiospastic types; also the typical, subtypical, and supratypical; and finally the ordinary type and the ophthalmic type. Practically we find two classes:

(1) The typical, associated with visual disorders and having most of the symptom described above.

(2) The irregular or mixed type, in which with many symptoms of ordinary migraine there is a history of rheumatic influences and often of anæmia or dyspepsia. These are cases of a true migrainous affection complicated with some form of symptomatic headache, such as has been already described. The mixed or irregular migraines are important to recognize, for they call for special treatment. They occur almost altogether in women; the attacks are often associated with weather changes and may be of a neuralgic character. Many women have their sick-headaches and their neuralgic headaches, so called, and have to distinguish between them. In mixed attacks the face is usually also pale, the eyes are not suffused, nor is there any visual or aural disturbance, as in migraine. Sometimes the pain remains in the occipital nerves, and I have met with one patient who localizes her pain there entirely, and who always vomits during the attack.

Pathology.—The seat of the disease is chiefly in the intracranial branches of the fifth nerve and of the pneumogastric; the upper cervical nerves, however, are often involved. There are no morbid anatomical changes known. The most plausible theory of the disease is that it is a fulgurating neurosis, in which there are periodical discharges of nerve force, or nerve storms. The seat of the dis-

charge is perhaps in the cerebral cortex, or possibly in the primary sensory centres, *i.e.*, the root ganglia of the fifth and vagus nerves. The disease is certainly not in the sympathetic system, as was once taught. The presence of excess of uric acid or of some other autochthonous poison as a factor in the disease may be regarded as probable.

Diagnosis.—The diagnosis is based upon the hereditary history, the periodicity and seat of the attacks, the nausea, the complicating visual and other sensory symptoms. It should not be forgotten that the same patient may have migraine and other neuralgias, or may have also an organic brain or renal disease.

Treatment—Prophylaxis.—Children of families in which this neurosis exists should be carefully watched during the ages between five and twenty. The eyes and nose should be examined. They should not be subjected to excessive mental or visual strain, and if attacks develop they should be promptly treated. The application of glasses should be considered, but not hastily adopted.

As regards constitutional treatment, the best measures for curing a case of migraine consist in correcting any visual or nasal defect, such diet and exercise as secure tone to the nerves, and the continuous use internally of bromide of potassium, nitroglycerin, cannabis indica, or arsenic. The diet should be very simple and non-fermentative. It should be mainly of meats and green vegetables and cooked fruits. The meats should be moderate in amount, and sometimes only fish and poultry should be allowed.

Of the drugs, salicylate of sodium and cannabis indica, either with or without arsenic, are the most trustworthy, while the bromides are the least. The hemp should be given in large doses and for a long time, gr. $\frac{1}{3}$ to gr. i., ter in die. The salicylates may be combined with an alkaline laxative like Rochelle salts, a dose being taken night and morning. Much stress is laid by some upon ocular muscular insufficiencies, and I believe that such conditions should be remedied, but place little confidence in them alone. On the other hand, the correction of small or large degrees of astigmatism and hypermetropia sometimes produces surprisingly good results. The reported cure of numerous cases of migraine by treatment of nasal hypertrophies and catarrh should not excite too much confidence in such measures. In fact, since migraine is a constitutional neurosis, one cannot expect permanent results from removing reflex irritants alone. The daily use of a strong galvanic current four to eight milliamperes (eight to ten cells) for ten minutes is useful.

For *the relief of the attack* the following drugs may be given: Salicylate of sodium, gr. xx.; caffeine, gr. ij. to v., with benzoate of sodium, gr. x.; antipyrin, gr. v. q. $\frac{1}{4}$ h.; phenacetin, gr. x. to xv.

or more; powdered guarana, gr. xx.; menthol, gr. v. to x. in hot water; muriate of ammonia, gr. xxx. to ʒ i.; bromide of potassium, gr. xx. Antipyrin and phenacetin are the most certain of the above drugs, but they lose their effect after a time, as do almost all the other drugs, and finally patients give up treatment or resort to codeine or morphine. Chloral and a hot foot bath break up attacks sometimes.

Locally galvanic currents are sometimes helpful, and so are static sparks. Hot applications and pencilling with menthol give relief to some. Quiet and rest are spontaneously resorted to.

Neuroses of the Acoustic Nerve.

Anatomy.—The auditory or eighth cranial nerve has two different parts. One portion passes to the cochlea and utricle and saccules; it has to do with the sense of hearing; the other goes to the semicircular canals, and has to do with that sense by which we appreciate the position of our body and its relations to space. The eighth nerve is thus an auditory and a space-sense nerve.

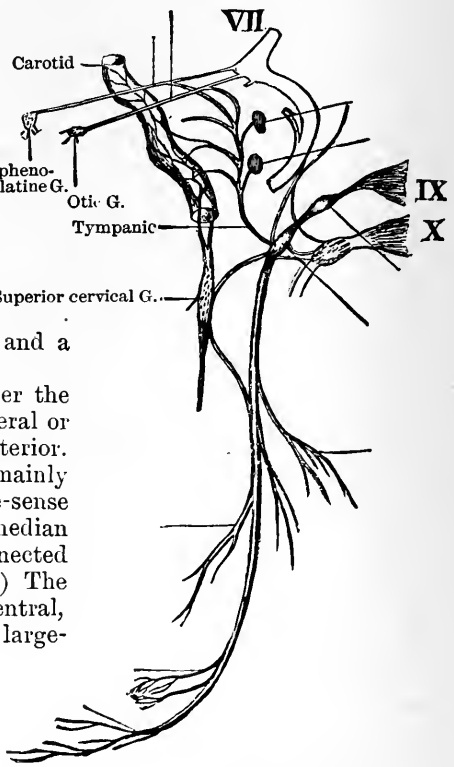


Fig. 98.—Glosso-Pharyngeal Nerve (Young).

The auditory fibres enter the medulla by two roots, a lateral or posterior and a median or anterior. The lateral root has mainly auditory fibres. The space-sense fibres enter chiefly by the median root. These roots are connected with three nuclei, viz.: (1) The chief nucleus (dorsal, central, inner nucleus); (2) the large-celled nucleus (Deiter's); and (3) the accessory nucleus (ventral, anterior, lateral). The chief nucleus (1) is a large mass of gray matter composed of small nerve cells and lies superficially just beneath the floor of the fourth ventricle. The large-celled nucleus (2) lies to the outer side of and below it. The accessory nucleus

(3) lies in the substance of the lateral root, and between it and the median root. The lateral root is the one coming chiefly from the cochlea, and is, as stated, mainly a nerve of hearing. It is connected most extensively with the accessory nucleus, but also with the other nuclei.

Through the accessory nucleus it connects by a few fibres with the superior olives, mostly of the opposite side; thence fibres pass up through the lateral lemniscus, to the posterior corpora quadrigemina; thence to the cortex of the first and second convolutions of the temporo-sphenoial lobes. Other fibres pass up directly through the lemniscus and the tegmental or sensory tracts to the cortex of the temporal lobes. Connections are numerous, also, with other cranial nerve nuclei. The lateral root also sends fibres to the chief nucleus and (*via* the striæ acusticæ) into the raphe, and thence to the formatio recticularis and sensory tract. These fibres go also to the posterior tubercles of the corpora quadrigemina and thence to the cortex of the temporal lobe.

The median root is connected chiefly with the large-cell nucleus (2), but also with the chief nucleus (1). From these nuclei fibres pass up in the posterior cerebellar peduncle through the direct sensory tract of the cerebellum to the roof nucleus or the emboliform and globose nuclei.

The diseases of the eighth or acoustic and space-sense nerve which are of special interest to the neurologist are auditory paralysis or nervous deafness, hyperacusis, tinnitus aurium, and auditory vertigo, or Ménière's disease. These diseases correspond with loss, excess, and perversion or irritation of function.

Nervous Deafness (Acoustic Paralysis).

Nervous deafness may be due to lesions (1) of the cortical centres of hearing, (2) of the acoustic nuclei, and (3) of the acoustic nerve itself or its end organ. Practically it is almost always the nerve and the internal ear which are affected.

Etiology.—1. Cortical nervous deafness has been known to occur in a few instances from lesions of both temporal lobes (Mills). A lesion in the left temporal lobe causes a form of sensory aphasia called word deafness. A lesion in the right temporal lobe may cause some deafness in the left ear. The deafness of hysteria is of cortical origin.

2. Deafness from lesion of the acoustic nucleus or nerve root.

Cerebro-spinal meningitis in the young and syphilitic meningitis in the adult are the more frequent causes of this form. Tumors and hemorrhages may also be causes. When the auditory nucleus and nerve root are affected by these diseases, its peripheral terminations in the labyrinth are also often involved, so that sharp dis-

tinctions cannot always be drawn beween this form and that due to labyrinthine disease.

3. *Deafness from Labyrinthine Disease.*—The causes are drugs, such as quinine and the salicylates; inflammations, including syphilitic exudates; injuries; hemorrhages; tumors; primary atrophy, which may occur in locomotor ataxia; mechanical causes, such as the constant noises and jarring to which locomotive engineers and boiler makers are subject.

Symptoms of Nervous Deafness.—The dominant symptom is loss of hearing, but this may be accompanied by vertigo, tinnitus, and even forced movements.

In hysterical deafness the loss of hearing is rarely complete, is unilateral, and especially involves high and low notes. Deafness from involvement of the nucleus and nerve root is usually accompanied by other symptoms of a basilar meningitis or lesion of the pons and medulla. Labyrinthine deafness is often associated with vertigo, tinnitus, and forced movements, when it may become a symptom complex known as "Ménière's disease."

Sudden total deafness is characteristic of syphilitic disease of the internal ear. In genuine nervous deafness, unless the deafness is absolute, bone conduction is lost, while aerial conduction is preserved. A tuning-fork vibrating on the skull or mastoid is not heard by the affected ear, though it is heard when held in the air close by this ear. Changes of reaction to the electrical current occur, but the tests are difficult and the results unsatisfactory.

The *treatment* depends upon the seat and nature of the lesion. In labyrinthine deafness it is generally limited to the use of iodide of potassium, mercury, pilocarpine, leeches, and the galvanic current. Local applications and surgical interference may be required. The treatment of nervous deafness in which vertigo, forced movements, and tinnitus are the dominant symptoms will be discussed under these heads.

Tinnitus Aurium. Tinnitus Cerebri (Noises in the Ear and Head).

Subjective sounds resembling hissing, buzzing, humming, beating, musical notes, etc., are classed together under the general head of tinnitus aurium. It is a very common symptom.

Etiology.—The disease attacks adults in middle and later life. Men and women are alike affected. Neuropathic constitutions and an unstable circulation favor it. The arterio-sclerosis of old age, cerebral anæmia and congestion, sunstroke, tobacco, and alcoholism lead to it. It occurs often in melancholia and in neurasthenia.

Some local disease or congestion of the middle or internal ear is usually present. Tinnitus occurs in Bright's disease, gout, and dyspepsia.

Disease of the auditory nuclei and auditory tracts rarely, if ever, causes tinnitus; but chronic pachymeningitis, such as follows blows on the head, sunstroke, alcoholism, etc., may be attended by most annoying tinnitus, which is often not so much in the ears as in the head—a tinnitus cerebri. In old people with thickened arteries and imperfect brain nutrition a similar condition may occur.

Tinnitus accompanies insanity sometimes, and may be the source of aural hallucinations.

A kind of tinnitus may accompany migraine and take the form of an aura in epilepsy.

Despite this long list of causes, the chief factors may be summed up as neurasthenic states, local ear disease, humoral poisons and irritants, reflex irritants, arterio-sclerosis.

The *symptoms* are indicated by the name of the malady. They may come on suddenly, but usually develop slowly. Some deafness and occasional vertigo are often present. The noise is located in one ear, as a rule. Sometimes it is said to be simply "in the head." The sounds are generally present all the time, giving the patient little rest and making life a burden. They vary greatly in character and intensity. These variations are indicated in the study of the diagnosis.

The Diagnosis.—The recognition of the symptom is easy. The principal thing is to discover its seat and cause.

The ear should of course be examined for external or middle-ear disease.

If the tinnitus is pulsating and synchronous with the heartbeats and stopped by carotid compression, it may be inferred that it is due to vasomotor paralysis, or inflammatory congestion, or aneurism.

If the sound is not in the ear, but in the head, and not associated with deafness or ear disease, the trouble is probably central, and most likely is of meningeal or arterio-sclerotic origin.

Noises which are complex or take the form of musical sounds or words are probably central.

Constant rushing, knocking, pulsating noises are due to congestion, hemorrhage, or inflammatory effusion in the labyrinth.

Moist sounds of a gurgling, bubbling, boiling, singing, whistling, shell-like roaring character indicate disease of the middle ear, with fluid exudation or catarrh of the Eustachian tube, or irritation of the external auditory canal, mastoid cells, or postnasal spaces.

Dry roaring and ringing noises are due to non-suppurative

catarrh of the middle ear, disease of the muscles or nervous supply of the tympanum, meningitis, tumors, and syphilis.

The condition of the digestion should be inquired into and the existence of renal, arterial, or central nervous disease investigated.

Treatment.—Hydrobromic acid and the other bromides, given in ordinary doses, are the surest remedies for this trouble. They may be combined with digitalis. Iodide of potassium and iodide of ethyl often are useful. Nitroglycerin is sometimes of value in patients with hard arteries. A combination of digitalis, bromide, and nitroglycerin has given me the best results. Occasionally tonics are indicated. I have seen but little good from electricity or counter-irritation. Of course middle-ear disease must be treated if it is present.

Vertigo (Dizziness, Giddiness).

Vertigo is a disturbance of consciousness characterized by apparent movements of external objects or of the person himself. If external objects whirl around, the vertigo is called *objective;* if the person himself seems to move, it is called *subjective.* Vertigo is almost always a symptom. In rare cases it appears to be *idiopathic.*

Vertigo is connected more or less with our space sensations, and hence it will be described here under disorders of the eighth cranial nerve and its central representations.

Etiology.—The causes of vertigo may be classed somewhat like those of headache, as follows:

1, Hæmic, etc., anæmia, hyperæmia, toxæmia from tobacco and alcohol; 2, arterio-sclerosis; 3, acoustic-nerve irritation; 4, neuroses; epilepsy, neurasthenia; 5, reflex: ocular, gastric; organic brain disease; 7, mechanical causes like electricity, swinging, etc.

Based mainly on the etiology, we have as a practical classification of ordinary cases of vertigo: toxic, auditory, gastric and bilious, ocular, neurasthenic, and epileptic forms. The various causes of vertigo act partly by irritating the space-sense nerve and thus disturbing our sense of relation to external objects, partly by irritating the cortical centres of the brain.

Symptoms.—Vertigo comes on suddenly, and lasts, as a rule, for but a moment. The floor rises and sinks, or objects whirl around (objective vertigo), or the patient seems whirling around or falling. The ideas are confused; there are a sense of alarm and a feeling of faintness. The patient totters, sometimes falls; there may be nausea or vomiting. In some forms there is momentary loss of consciousness, or syncope. Vertigo usually comes on in short attacks, but in toxæmic states, as in alcoholism or nicotinism, it is

almost constant while the poison is in the system. Vertigo may become chronic or nearly so; and if severe it forms what is called the *status vertiginosus* (Mitchell). Vertigo is increased by rising or sudden movements and lessened by lying down.

Vertigo may be due to organic lesions of the cerebellum and its peduncles, or of the labyrinth; it is then associated with forced movements of the body.

Symptoms of Special Forms—Auditory Vertigo (Ménière's Disease).—A large proportion of vertigoes are due to disease or irritation of the eighth nerve and its centres. The common cause is local disease of the labyrinth. When this produces severe attacks of vertigo with nausea and perhaps syncope, it is called "Ménière's disease." The name is often applied to any form of auditory vertigo. Ménière's type is always due to organic disease of the labyrinth. It is accompanied by progressive deafness, and sometimes by tinnitus and forced movements, or even an utter inability to walk steadily. When the deafness is complete the vertigo ceases, because the nerve end organ is destroyed. Mild forms of auditory vertigo present nothing unusual except those due to involvement of the nerve of hearing.

Many forms of vertigo, such as the gastric and toxic, occur through a reflex disturbance of the eighth nerve. The auditory nuclei are connected with those of the vagi. The labyrinth is supplied with blood by the vertebral artery, whose calibre is controlled by sympathetic fibres which are in close connection with fibres to the stomach. Hence reflex effects may occur through contiguity of the central nuclei and by reflex spasm of the vessels of the internal ear. "Stomachal vertigo" is the name given to a very severe form of reflex vertigo. It occurs generally in persons whose stomachs are overloaded and whose digestion is paralyzed by its load. It is accompanied by loss of consciousness.

Bilious and Lithæmic Vertigo.—In conditions of dyspepsia, constipation, and hepatic torpor, the disordered stomach and bowel suddenly discharge into the blood irritant substances which pass to the brain and by direct action on the nervous centres cause vertigo. This is probably the explanation of the vertigo of biliousness and constipation. It is a paroxysmal vertigo, noted most in the morning, not very severe, and often accompanied by nausea.

Neurotic Vertigo.—The symptoms of epileptic vertigo will be described under that head.

Neurasthenic vertigo is a not uncommon symptom. The attacks are short, generally subjective, not severe or accompanied by nausea or syncope, but they often cause much alarm. Underlying them are

exhausted and irritable nerve centres, with ocular, gastric, and humoral irritations.

A neurotic vertigo occurs sometimes in the form of attacks almost exactly resembling seasickness. There are intense vertigo, nausea, and faintness lasting for hours, coming on suddenly without known cause except overwork or excitement. The attacks occur in neurotic subjects and are analogous to other nervous crises. It is a periodical neurosis of the space-sense nerve.

A form of vertigo which is *psychical* in character occurs in neurasthenics. It consists in a sudden sensation of insecurity, an apprehension of falling, of an approaching loss of consciousness. There is no true vertigo, either subjective or objective, and the patients really never stagger or fall. It is a psychosis rather than a nervous condition.

In some nervous subjects there occurs a sudden giving way of the legs. There is no conscious vertigo, yet such probably exists. The symptom is noted in exophthalmic goitre. It is a "stumbling vertigo."

Ocular Vertigo is a rare symptom, but is, when present, chronic and annoying. It is caused by refractive errors and unequal action of the ocular muscles.

The *mechanical vertigoes* such as seasickness, car sickness, etc., are produced by swinging, or whirling, the movements of the ship, steam car, and elevators. Railway mail clerks, elevator boys, often suffer from chronic disturbances of a vertiginous character. Ocular and auditory nerve sensations enter mainly into the causation of the troubles.

Arterio-sclerotic Vertigo, Senile Vertigo.—This occurs in persons who have arterio-sclerotic changes in the brain vessels, either from disease or senility. The symptom is caused by impaired brain nutrition with consequent anæmia. Senile vertigo may also be due to a weak and fatty heart.

Pathology.—The consciousness of the proper equilibrium of the body and of its relations to the external world depends upon the continuous inflow of nervous impulses from the eye and its muscles, from the nerves of the muscles, joints, and viscera, and from the ear. Anything which suddenly disorders this even inflow may cause a disturbance of consciousness and sensations of vertigo.

The aural impulses come from the semicircular canals and ampullæ; they are the most important. These impulses are not felt in consciousness normally, but go to certain lower centres chiefly in the vermis of the cerebellum. From this point they influence the acts concerned in holding the body in equilibrium.

When impulses from the eye and its adjusting mechanism do not flow in normally, there may be disturbance of consciousness and a feeling of vertigo. Probably visceral impulses can produce a similar disturbance. Everything which suddenly interferes with the nutrition of the cortex of the brain, such as anæmia and poisons, may lead to giddiness by lowering the level of consciousness and confusing the sensory inflow.

Diagnosis.—In investigating vertigo the physician should find (1) whether it is subjective or objective, (2) paroxysmal or chronic, (3) accompanied by ear symptom, nausea, tinnitus, and loss of consciousness. He should then direct himself to finding the special cause and seat, remembering that the auditory, gastric, toxic, and neurasthenic are the common forms. In elderly persons the arteries should be carefully examined. In young persons the possibility of epilepsy must be remembered.

The *prognosis* depends upon the cause. Epileptic vertigo and vertigo from organic disease are most serious. Labyrinthine vertigo usually ceases when complete deafness occurs. The other forms of vertigo are usually susceptible of relief.

Treatment.—The attack is treated by rest in the horizontal position and the administration of a volatile stimulant. The disorder must then be treated in accordance with the cause.

In Ménière's vertigo the use of quinine by Charcot's method is said to be useful. Quinine is given in doses which are gradually increased until cinchonism results; then the drug is stopped. Mitchell advises the addition of hydrobromic acid; Gowers advises the use of salicylate of sodium in five-grain doses instead of quinine. Hirt recommends ten drops of a two-per-cent solution of pilocarpine injected hypodermically every other day.

Neurasthenic vertigo is cured by rest and attention to diet, laxatives and mineral acids being used. Hydrobromic acid with pepsin and glycerin are often very helpful here. Gastric vertigo is to be treated with saline laxatives and simple bitters before meals.

In the vertigo of "biliousness" and lithæmia there is often a neurasthenic element, and a similar attention to diet and to the digestive organs is indicated. In arterio-sclerotic and senile vertigo small doses of nitroglycerin and iodide of potassium, with or without digitalis, should be given. Rest and warmth of the extremities are indicated. In all forms of vertigo bromide of potassium is helpful and will relieve the symptoms for a time. It is the best symptomatic remedy. Counter-irritation to the neck or mastoid region by the cautery does good occasionally.

There are two peculiar forms of disease to which the name vertigo has been attached which may be described here.

Laryngeal Syncope (Laryngeal Vertigo, L. Epilepsy).—This is a rare form of disorder characterized by attacks of paræsthesia of the throat, with coughing, followed by sudden syncope, and sometimes by slight convulsive movements.

The disease occurs chiefly in males at about the age of fifty, though the range of age is from thirty-five to seventy. Neurotic constitution is often present. There may be a history of injury and the use of stimulants. Laryngitis, bronchitis, apical phthisis, and asthma may be present.

At the onset of the attacks a burning or tickling sensation is felt in the larynx or trachea; there is a spasmodic cough, perhaps some asthmatic or dyspnœic symptoms, when the patient suddenly falls unconscious for a short time. The attacks may occur daily or only once in a few weeks. Most cases are curable, yet the disease is not without danger.

It is probably a reflex neurosis, not a true epilepsy.

The treatment should be directed to relieving any local condition or pulmonary trouble. Bromide of potassium should also be given.

Paralyzing Vertigo (Gerlier's Disease).—This is a disease occurring only on the farms in southern France and Switzerland.

The symptoms consist of sudden attacks of ptosis, vertigo, paresis of arms and legs, and cervico-occipital pain. The disease is most prevalent in the summertime. It attacks chiefly males. Single attacks last not over ten minutes, but may occur frequently.

The cause is supposed to be a special microbe developed in the stables during the heat of summer.

We are not aware of its occurrence in America, though Seguin calls attention to the close similarity of the symptoms to those of poisoning by conium maculatum.

Hyperacusis (Auditory Hyperæsthesia).—When there is undue keenness of the sense of hearing, the condition is called hyperacusis. It occurs in hysteria and hypnotic states. Some persons have naturally an extraordinary keenness of hearing. In facial paralysis there is sometimes hyperacusis due to paralysis of the stapedius.

When ordinary sounds cause painful feelings, the condition is called dysacusis. This occurs in the neurasthenic and hysterical, in persons of enfeebled vitality, in the brain congestion of fevers, and in meningitis; also in local ear troubles of an inflammatory character.

Sensory Neuroses of the Glosso-Pharyngeal Nerve.

The anatomy of this mixed nerve is described under the head of motor neuroses. The sensory fibres may be affected in hysteria, causing the symptom called globus, and also the pharyngeal anæsthesia found in the same disease.

The special fibres of taste may be affected, causing ageusia or loss of taste.

Ageusia (loss of the sense of taste) is an affection in which the power to discriminate the tastes of bitter, sweet, salt, acid, and alkaline substances is lost.

Etiology.—It occurs oftenest in an incomplete form in facial palsy and in hysteria. Injuries of the trigeminus and glosso-pharyngeal nerves, catarrhal diseases of the mucous membrane of the mouth and nose, are frequent causes. It is not caused by cortical brain disease so far as known.

Some ageusia is present in the imbecile, and the sense of taste is less keen in the lowly organized and criminal classes.

Symptoms.—The symptoms are subjective and may not be noticed at first by the patient. In hemiageusia from facial palsy and hysteria it has to be looked for, as the patient does not complain. The tests are made with solutions of salt, sugar, vinegar, and quinine. A single solution of sugar usually answers. But the different parts of the tongue differ in sensibility to different substances (see Fig. 97). Care must be taken to exclude the nose as a factor in taste.

Pathology.—Ageusia occurs as the result of disease of the roots of the trigeminus, especially of the second, or, as Krause claims, the third; also from disease of the facial when the chorda tympani is implicated and from disease of the glosso-pharyngeal root.

Disease of the trigeminus and facial usually causes ageusia on the anterior two-thirds of the tongue, with loss of taste, especially for sour and bitter substances. Sometimes, however, disease of the trigeminus or disease of the tympanum involving the tympanic plexus and chorda tympani causes ageusia of the whole tongue on the affected side.

Ageusia from disease of the glosso-pharyngeal alone is very rare, and then causes loss of taste on the posterior third of the tongue, soft palate, and pillars of the fauces, with loss of taste to sweets and acids. A few cases have been reported in which paralysis of the glosso-pharyngeal caused complete ageusia on the affected side. It must be inferred, therefore, that taste fibres run sometimes wholly in the fifth, more rarely wholly in the ninth nerves, and usually in both.

The *treatment* depends on the cause. Locally, cleansing and stimulating mouth washes and electricity may be used.

Parageusia, or perversions and imperfections in the taste sense, are very frequent. They are generally due to irritation of the taste nerves from catarrhal inflammation of the stomach or mouth. They also occur in hysteria.

Sensory Neuroses of the Upper Cervical Nerves.

The anatomy of these nerves has been described under the motor neuroses. The sensory distribution to the skin is shown in the accompanying figure (Fig. 98).

Cervico-Occipital Neuralgia — Neck Pains — *Etiology.*— Pains in the back of the head and neck occur in migraine, in hysteria, spinal irritation, and neurasthenia, as a result of eye strain, as a true neuralgia, and as a symptom of brain tumor, meningitis, and rheumatic inflammation of the neck muscles and nerves. True cervico-occipital neuralgia is not rare, is much more common in women, occurs between the ages of twenty and thirty-five, and is often a reflex of pelvic disease.

Symptoms.—Migrainous pains are described elsewhere. In spinal irritation and hysteria the trouble is central, or perhaps shifting; it is especially characterized by a sharp boring pain just below the occiput. With it there may be evidences of cerebral congestion or anæmia, with vertigo and faintness, but not vomiting. The boring pain is almost pathognomonic of spinal irritation. In neurasthenia the pain is more of a tired, aching character. In typical neuralgia the pain is usually unilateral, paroxysmal, and sharp, sometimes reaching the intensity of a tic douloureux. There are tender points over the exit of the nerves. The disease lasts for five or six weeks. If of reflex origin, it may become chronic. The pains may alternate with or take the place of a trigeminal neuralgia. The nerves involved are the great and small occipital from the second pair and a branch from the third pair.

Treatment.—General constitutional treatment consists in the use of antirheumatics, such as the salicylates. The muriate of ammonia has done me good service. In women pelvic troubles should be looked for; in both sexes the eyes must be attended to. Locally, counter-irritants, cupping, and leeching are useful; mustard and capsicum pastes are often a great relief. The ice bag also is of service. Trephining the occipital bone has cured one obstinate case, and resecting the occipital nerves another.

Sensory Neuroses of the Lower Cervical and Brachial Nerves.

Cervico-Brachial Neuralgia.—The cutaneous distribution of the sensory nerves of the arm is shown in Figs. 81 and 98. Cervico-brachial neuralgia is relatively rare; it occurs oftener in

women and in early adult and middle life. The ordinary causes of neuralgia produce it, but rheumatism and gout are rather prominent factors. Overuse of the arm, in anæmic neurasthenic persons, is a most potent cause. Reflex irritation from carious teeth and uterine disease have been found to be causes. It occurs symptomatically in locomotor ataxia and other cord diseases. A low grade of neuritis probably often exits.

Symptoms.—It begins somewhat gradually with aching pains in the neck, shoulder, axilla, and along the course of the nerves. The pains gradually increase and are usually worse at night. The pain is increased by use of the arm and by exposure. Only one arm is affected. Painful points may be felt in the axilla, at the lower end of the scapula, over the deltoid, over the ulnar near the wrist, over the lower part of the radial and sometimes on each side of the lower cervical vertebræ. There are usually paræsthesias and numb feelings. If there is a complicating neuritis, burning sensations are felt ("causalgia"). There may be anæsthesia, vasomotor disturbances, herpes, and muscular weakness and atrophy. Brachial neuralgia generally involves all the nerves of the plexus. If special nerves are involved the ulnar is oftenest affected, next the musculospiral, and last the median.

Digital neuralgia sometimes occurs. The pain is often located in a single finger. The cause is usually a local injury or neuritis. Sometimes it is a reflex pain due to some remote trouble. In a few cases this remote trouble is uterine.

Diagnosis.—The etiological diagnosis is most important. Inquiries as to a rheumatic or gouty element should be made. The presence of any serious amount of neuritis would be shown by local tenderness and motor weakness. Organic cord disease must be excluded.

The *prognosis*, if the neuralgia is functional, is good. If a neuritis complicates it, it is more serious. If the neuritis, however, is slight and is secondary to a trauma, or is rheumatic or gouty, the prognosis is favorable. Ordinary attacks last about six weeks.

The Treatment.—Salicylate of potassium, iodide of potassium, or muriate of ammonia should be given and in large doses. Aconitia and gelsemium are not of much value. The electrical current, either faradic or galvanic, has a very decided effect for good. Leeching helps some cases. Blisters may be used in neuritic cases. The analgesic drugs like phenacetin and antipyrin and antifebrin are useful symptomatically. Hot local applications often give some relief. Colchicum cures some cases of gouty origin. The most important thing of all, however, is to secure rest for the arm and also

for the patient. Putting the patient to bed is the best medicine; and often the arm needs to be put in a splint or sling.

The Sensory Neuroses of the Intercostal Nerves.

These are chiefly intercostal neuralgia, symptomatic side pains, herpes zoster. Paralyses and anæsthesias occur in connection with vertebral and spinal-cord diseases.

Intercostal Neuralgia; Side Pains—*Etiology.*—This is a very common neuralgia. It occurs much oftener in women (partly from corset pressure) than in men (seven to one). The favorite age is twenty to thirty-five; the season, winter. Anæmia, neurasthenic and hysterical conditions, child bearing, pelvic disorder, dyspepsia, heart disease, malaria, and lead poisoning are frequent causes. Exposure and muscular strain are rare exciting causes.

Symptoms.—The disease comes on suddenly. The pain in typical cases is sharp and stabbing, but not much increased by respiratory movements. There are tender points at the seat of pain, which is usually greatest over the side at the exit of the lateral nerve branches. Often a tender point is felt over the exit of the dorsal or of the anterior branch. It is rare to find all three points. The disease runs about the course of facial neuralgias, *i.e.*, of two to six weeks, but it is sometimes very obstinate, lasting for many months. The sixth to tenth nerves are those oftenest involved. The left side is more susceptible than the right (three to one).

Pathology.—In most cases the nerve is irritated by poor or poisoned blood. In a minority of cases the pain is reflex from stomach, pelvis, or heart. Sometimes there is a neuritis.

Diagnosis.—Probably one-half the pains in the side are myalgic in nature, and should be classed as pleurodynia. These pains can be distinguished by the history of their origin and of rheumatic influences, by their diffuseness and dulness, by the great tenderness on pressure, and the pain produced on taking a deep breath. There is another considerable proportion of cases in which the pains are mainly neuralgic, but yet there are some evidences of muscular complications. Some of these pains are reflex.

In the third class of cases there is the pure stabbing neuralgia. The diagnosis is based on the exclusion of pleurisy, rheumatic and reflex causes, by the character of the pain, and the presence of tender points.

Prognosis.—The prognosis is good, except for a few chronic cases due probably to a degenerative neuritis. In some of these cases there is lead poisoning.

Treatment.—The most efficacious treatment is a blister and ferruginous tonics with quinine. In all cases the heart, pleura, stomach, and pelvic organs must be examined, and any disorder relieved. Iodide of potassium and chlorodyne have cured some very bad cases in my experience. If there is a rheumatic and muscular element, give salicylates or the analgesics, apply heat, and secure rest by adhesive straps.

Mammary neuralgia (mastodynia) is a form of intercostal neuralgia involving the anterior and lateral branches of the three or four upper dorsal nerves.

Etiology.—It is caused by local tumors, or it may be an essential neuralgia. The causes in the latter class are anæmia, pendent breasts, pressure from badly fitting corsets, and injury.

Mammary neuralgia also occurs in hysterical women and young girls sexually precocious; it may occur in pregnancy and during lactation. Many mammary pains are due simply to local disorder of the gland.

Symptoms.—Mammary neuralgia is unilateral, often very severe, and if it occurs in middle life is liable to cause much mental depression from fear of cancer.

The *treatment* depends upon the cause. It requires general tonic measures and attention to the proper support and protection of the gland.

Herpes Zoster, Dermatitic Neuritis (*Shingles*).—Almost the only recognized form of neuritis of the intercostal nerves is known under the name of *herpes zoster*. This is an acute dermatitis, secondary to the neuritis.

Etiology.—Its predisposing causes are wounds, the morphine habit, rheumatic, gouty, and syphilitic poisons, and emotional influences. The active cause is in all cases an infection, and the disease sometimes is almost epidemic. The inflammation affects not only the nerves but the spinal ganglia, and especially the latter, so that the condition might be called a posterior poliomyelitis (Head).

Symptoms.—It begins gradually with the development of pain and a herpetic eruption upon one side of the trunk. It generally involves the lower dorsal nerves. The eruption follows the course of the nerve, rarely extending to the opposite side. The pain gradually subsides, and the disease itself runs its course in a few weeks.

Treatment.—In the early stage the galvanic current is efficacious. Local anodynes and protective ointments may be applied. Antirheumatics and analgesics are to be administered internally.

Sensory Neuroses of the Lumbar Nerves.

The upper two lumbar nerves are almost entirely sensory. Neuralgias of these nerves are called lumbo-abdominal.

They occur oftener in women and usually after the thirtieth year. To the ordinary causes of neuralgia we add straining, constipation, and pelvic disease. True essential neuralgia is rare; but myalgic and reflex pains from uterine disease are very common.

Symptoms.—There is pain in the loins, back, and buttocks, extending down to the hypogastrium or genitals on one side. The pain in the back, however, is often bilateral. Painful points may be found after a time, as in intercostal neuralgia. Sometimes the pain is located in the side of the penis (penile neuralgia). Neuralgia of the long lumbar branches is called femoral or crural. A common form of this constitutes what is called *painful thigh.* When these nerves are subject to a lesser irritation, causing sensations of numbness and pricking along the thigh, the condition is called *meralgia.* In true neuralgia, the patient complains of pain in the front of the knee and the anterior and outer parts of the thigh, but has no pain posteriorly and none below the knee. The internal branches of the anterior crural nerve do not seem to be affected, while the middle and external cutaneous branches and the genito-crural nerve are involved.

Disease of the hip or of the sacro-iliac joint or vertebræ may cause a reflex pain in the obturator nerve, localized especially in the knee and back of this joint. The fœtal head sometimes compresses these branches, causing a symptomatic neuralgia. Diseases of the internal genital organs are especially liable to cause reflex pain in the lumbar nerves. Diseases of the external genitals and bladder more often reflect pains upon the sacral nerves. In biliary colic pains are felt in the ilio-inguinal and hypogastric nerves. Local disease of the psoas muscle or iliacus, in the neighborhood of Poupart's ligament, causes pains in the lumbar nerves. Lumbar neuralgias generally run a favorable and not very long course.

Diagnosis.—Lumbar neuralgia is distinguished from lumbago by the unilateral position, the distribution and paroxysmal character of the pain, and the lack of severe suffering on motion and pressure; the tender points and the absence of any organic disease. Lumbago comes on suddenly, with a history of exposure, is bilateral and confined to a single group of muscles, which are tender on deep pressure. In lumbar sprain the onset is also sudden, with a history of injury, great local tenderness, and evidences of trauma.

The *treatment* is the same as that for neuralgia in general. The

frequent presence of pelvic disease and of anæmia and a rheumatic history must be borne in mind.

Sciatica (Neuralgia of the Sciatic Nerve, Sciatic Neuritis).—This is a form of neuralgia occurring in middle life and characterized by intense pain in the course of the sciatic nerve. A large proportion of the cases is due to a neuritis.

Etiology.—The disease occurs three times as often in men as in women, and is the only neuralgia of which this can be said. Most cases in this country occur between the ages of forty and fifty; next between thirty and forty.*

The gouty and arthritic diathesis, and occupations which lead to exposure and strain, predispose to the disease. It is not rare, therefore, among laboring men. In younger persons a neurotic constitution predisposes to the disease, and in this class the trouble is more truly of a neuralgic character and less of a neuritis.

Most cases occur in the autumn and winter. The exciting causes are constipation, pressure from hard seats, exposure, muscular strain from heavy work, and pelvic disorders. Symptomatic sciatica may be caused by the pressure of pelvic tumors, injury to the nerves, inflammation, vertebral and spinal disease; sciatica occurs in diabetes and in phthisis. In elderly persons of a rheumatic constitution inflammatory processes about the hip-joint complicate or cause the neuralgia.

Symptoms.—The disease begins rather suddenly. Pain is felt in the back of the thigh, running down the leg in the course of the nerve. Generally it is most marked in the thigh, extending up often into the lumbar region. Sometimes the disease begins like a lumbago; more rarely pain is first felt in the calf or foot. The pain is increased by motion, and the patient holds himself in a constrained position. The pelvis is tilted up toward the sound side and the trunk inclined over to the diseased side. After a time this leads in some cases to a characteristic deformity (sciatic scoliosis) in which the convexity of the curve of the vertebral spines is directed toward the diseased side. The pain is almost continuous, with paroxysms of great severity, which often occur at night. During these paroxysms the pain is sharp, burning, and lancinating. In the interval it is dull. Besides the pains the patient suffers from feelings of numbness, tingling, and a sense of coldness and weight in the affected limb. There are almost always tender points over the course of the nerve. These may be found at the sciatic notch,

* Personal statistics (102 cases) and those of Dr. L. Putzel (53 cases) give: Males, 111; females, 44. Ages: 10–20, 4; 21–30, 30; 31–40, 43; 41–50, 44; 51–60, 18; 61–70, 12; 71–80, 4.

at the middle of the hip, behind the knee, just below the head of the fibula in the middle of the calf, behind the external malleolus, and on the back of the foot (Fig. 99).

A pain running up the back of the thigh may be caused by pressure over the back of the knee when the leg is extended at a little more than a right angle. This is diagnostic (Gowers). If the patient lies on his back and the leg is kept extended, and then the whole limb brought slowly up until it is at an acute angle with the trunk, a sharp pain in the sciatic notch is felt; this too is diagnostic. Anæsthesia over the course of the nerves occurs very rarely. When present, it indicates a severe neuritis or injury to the nerve. Muscular wasting and weakness occur after a time, and in old and severe

FIG. 99.—SHOWING THE TENDER POINTS IN SCIATICA.

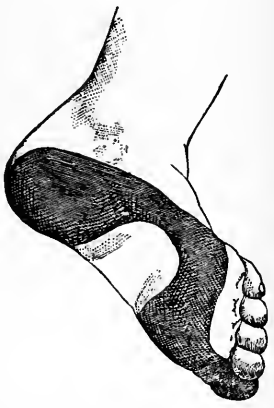


FIG. 100.—PLANTAR FORM OF SCIATICA; PLANTAR NEURITIS, showing area of pain (Mitchell).

cases partial electrical degeneration reactions may be observed. Herpetic eruptions over the course of the nerve occur in rare cases. The affected limb usually feels colder and shows evidence of enfeebled vasomotor supply.

The disease usually lasts two or three months; not rarely it lasts six months or even a year or more. It has been known to extend slowly upward and involve the sacral plexus or even the spinal cord.

Pathology.—The trouble is, as already stated, a chronic perineuritis in the majority of cases.

Diagnosis.—Sciatica has to be distinguished from hip-joint disease, organic disease of the cauda equina or cord, muscular pains in the hip and leg, and from pains caused by tumors. Pure sciatic neuralgia ought also to be distinguished from sciatic neuritis. A

consideration of the facts already given ought to make the diagnosis not difficult. Pure sciatic neuralgia occurs in early life and is not accompanied by much local tenderness. There is no paralysis or wasting of the limb. Double sciatica is most always symptomatic of diabetes or organic disease. True sciatica is rarely double.

Prognosis.—Almost all cases get well in from three to six months. Severe attacks in people over forty are the most intractable. Relapses occur, but not as a rule.

Treatment.—In all cases which are seen early, the most important indication is rest. The patient should be put to bed, and the whole lower extremity secured in a Thomas splint extending from ankle to axilla. Ice bags or linseed poultices or leeches should then be applied over the course of the nerve. A blue pill (gr. v.) may be given twice daily at first. In less severe or older cases large blisters should be applied over the nerve in the thigh, and the application repeated in a week. If there is a rheumatic history, potassium salicylate or iodide should be given in full doses. The bowels must be freely opened. Hypodermic injections of morphine or cocaine (gr. $\frac{1}{4}$) may be needed for a few days, the cocaine being repeated if necessary. When the disease has become more chronic a strong galvanic current may be given daily with large electrodes, one over the lumbar region or sciatic notch, the other, which should be the positive pole, over the leg and foot. As so-called specific remedies we have oil of turpentine in doses of fifteen drops t. i. d., and this may be advantageously combined with oil of gaultheria. Massive doses of antifebrin or antipyrin sometimes stop the pains (gr. x., q. 2 h.). There are a great many local remedies which at times prove useful. Among these mustard plasters, menthol, chloroform liniment, setons, acupuncture, cups, and the actual cautery and chloride of methyl can be recommended. Bandaging the limb in sulphur to which a little menthol is added is often very efficacious. Kneading the nerve with a glass rod and an anodyne ointment is sometimes beneficial. Very little can be expected from nerve-stretching, but it may be tried as a last resort. If tried, however, great caution should be exercised in pulling on the nerve. Not over thirty to forty pounds pull should be used. The operation of cutting down on the nerve and dissecting off the sheath for a space of several inches may be tried.

Plantar Neuralgia.—In rare cases the pain of sciatic neuralgia is limited to the plantar nerves, and is accompanied by paræsthesia and even anæsthesia of this region. The condition here is probably a neuritis combined sometimes with arthritic changes. Erythromelalgia may be regarded as a form of plantar neuralgia.

Erythromelalgia (*red neuralgia of the feet, congestive neuralgia*) is a disease affecting the feet chiefly, and characterized by burning pains and congestion of the parts.

The disease occurs usually in men in middle life, after some fever or severe physical exertion afoot. It is due sometimes to gouty habits, and I have seen it in patients who were diabetics.

The disease begins in the ball of the foot or the heel with burning pains. The trouble increases until nearly the whole sole in the distribution of the plantar nerve is involved, and the pain, though worse at night, is almost continuous. It is much increased by exertion, the feet become very tender, and standing or walking is most painful. Meanwhile there has developed with the pain a flushing of the part upon exertion. In bad cases the parts most affected are continuously marked by a dull, dusky, mottled redness, with some swelling. The hands may be slightly affected. Slight injuries may cause blisters and even ulcerations. The congestion usually disappears in the horizontal position, and this also relieves the pain. The symptoms are worse in warm weather. The disease is very chronic and, though not dangerous to life, makes life very miserable.

Pathology.—In the cases of erythromelalgia as described by Mitchell and others, there are: (1) A vasomotor disturbance; (2) a neuritis; and (3) in rare instances, spinal-cord disease; (4) sometimes there is an obliterative arteritis. The condition in my experience is closely associated with diabetes.

The *diagnosis* must be made from alcoholic and gouty paræsthesia, podalgia, local disease of bone and ligaments, and from reflex pains.

Treatment.—Elevation of the feet and applications of cold give temporary relief. Faradization has sometimes given help; oftener it has not. There is nothing known which gives permanent relief. The physician must rely upon rest, bandaging, cold, anodyne applications, hydrotherapy, and tonics. The salicylates, turpentine, and mineral acids with strychnine may be given. Benefit in lithæmic cases is obtained from codeine and an antidiabetic diet.

Plantar Neuroses of Mechanical Origin.

Morton's neuralgia, so called, is a neuralgia affecting the metatarso-phalangeal joint of the third and fourth toes, and is due, it is thought, to a slight luxation, with consequent pressure on a digital branch of the external plantar nerve. It sometimes affects other toes, however. It is not always due to a luxation. Incipient flatfoot may cause it, and I have seen a typical case in a pregnant woman, disappearing after confinement. The trouble occurs generally in women, and if there is a luxation the cause is external injury

or shoe pressure. The treatment is not very satisfactory. It should be directed to giving rest to the foot, and the avoidance of lateral pressure on the joints by wearing a broad-soled shoe with support to the arch of the foot. Support may also be given by a broad flannel bandage. Amputation of the toe is a very certain remedy.

Tarsalgia (*policeman's disease*) is a neuralgic affection, due probably in most cases to an incipient flattening of the foot and stretching of the plantar ligaments. Some have ascribed it to a deep-seated contusion of the adipose cushion covering the os calcis. A chronic inflammation of the sheath of the tendo Achillis causes symptoms resembling podalgia. Probably the condition varies somewhat in different cases. It is observed in persons who have been in the habit of going barefoot, and have then gone into the army or taken civil positions obliging them to stand or walk a great deal.

It was noticed originally in the policemen of Paris, and cases have been seen in this country. The name tarsalgia was given by Duchenne.

Treatment, medical or surgical, seems to do little for the disorder, which is very chronic. Patients are better in cold weather, and when resting the feet. Leeches, the cautery, the iodides, and broad shoes with rubber heels are serviceable.

Coccygodynia is a neuralgia affecting the lower posterior branches of the sacral nerves. It occurs oftenest in women and is caused by exposure, injury, and labor. Coccygeal pains occur in spinal irritation and reflexly from pelvic disease. The disease is a most annoying one, as it interferes with sitting and walking. There is often also pain at stool, and the parts are tender to pressure. The disease is usually one involving the fibrous structures of the coccyx, and is more an articular and bony than a nervous disorder. Surgical treatment, such as amputation of the coccyx, may be needed, and is sometimes effective, but not in the cases in which there is only a neurasthenia with spinal irritation.

Peripheral Vasomotor and Trophic Neuroses.

Symmetrical Angio-Neurotic Gangrene, or Raynaud's Disease (Abortive Form Known as Digiti Mortui).—Symmetrical gangrene or Raynaud's disease is a rare affection characterized by spasm of the vessels of the extremities, coldness, pallor, waxiness of fingers or toes, or by blueness, mottling, swelling, pain, followed often by a dry gangrene of some of the fingers or toes.

Etiology.—The disease occurs usually in children and young adults. Women are affected oftener than men. Anæmia, and chlorosis, and neurasthenic states predispose to it. Malarial infection, acute infectious fevers, menstrual disorders, fright, occupations that lead to exposure, such as washing, are causative factors. Diabetes and syphilis are also put down as causes.

Symptoms.—The disease comes on rather suddenly and affects oftenest two or three fingers of both hands. In its early and mild degree there are simply a coldness, numbness, and waxy pallor of the fingers. The skin looks shrunken. There is slight anæsthesia.

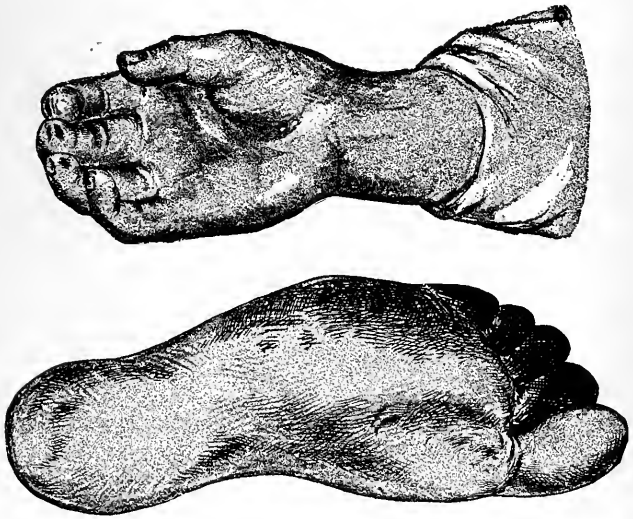


Fig. 101.—Hand and Foot in the Gangrenous Stage of Raynaud's Disease.

The extremities feel as if dead. After a few hours this passes away, but returns again and may finally become an almost constant condition. Beginning in one or two fingers, it may finally involve all. The toes, tip of the nose, and ears may be similarly affected, though this is rare in the milder form. Exposure to cold, even slight, is the common excitant of this form of the trouble, which is commonly known as "digiti mortui," "dead fingers," or "local syncope."

In severer grades the fingers become blue, swollen, and there are burning sensations and much pain, but no anæsthesia. This condition is known as that of "local asphyxia," and it is usually followed by gangrene.

In the gangrenous stage small blisters appear on the distal phalanges, which fill with bloody serum, then dry up, and beneath

the scab ulceration begins, which is shallow and soon heals, leaving a scar. The process then stops. In very rare cases the whole tip of the finger or toe, including the bone, becomes involved. The process as stated may attack the ears, lips, tongue, and even parts of the trunk. Along with this gangrenous process there is often a hæmaturia.

The dead-finger trouble may last but a few days or weeks, or it may continue for months. The gangrenous stage lasts about three weeks. It lasts longer if it comes on in one finger after the other. The disease is one of months, and it is liable to recur.

The *diagnosis* must be made from senile gangrene, frostbite, ergot poisoning, alcoholic neuritis, endarteritis, and obstruction of nutrient vessels.

Pathology.—A neuritis has been found in some cases of so-called Raynaud's disease, but this is secondary. In a few other cases there has been found an obliterating endarteritis (Jacoby). The trouble is in some cases apparently functional and due to the combination of an oversensitive nervous system and some irritant, such as impoverished blood, malaria, or other toxic agent, which causes spasm of the peripheral vessels.

Prognosis.—The cases usually get well, but the course is long and there may be relapses. In only the rarest instances has death occurred, and then from some complication.

Treatment.—Galvanism to the spine and limbs, warm applications, anodynes, tonics, are indicated. Nitroglycerin, the iodides, chloral may be tried. No specific is known.

CHAPTER XI.

DISEASES OF THE SPINAL CORD.

Anatomy and Physiology.

Anatomy.—The spinal cord is a slender, cylindrically shaped organ. It is from 42 to 45.7 cm. (sixteen and one-half to eighteen inches) long, being shorter absolutely and relatively in women. Its weight is about thirty-three grams (one ounce). It is suspended in the vertebral canal, where it reaches in all persons over one year of age as far down as the second lumbar vertebra. In new-born infants it extends to the third lumbar vertebra.

It is divided into cervical, dorsal or thoracic, lumbar, and sacral portions, corresponding with the nerves it gives off. These are respectively four, ten and one-half, two, and one and one-half inches long (see Fig. 102). Its shape on cross-section is nearly round, except in the lower cervical region, where it is flattened antero-posteriorly. Its average diameter is 1 cm. (two-fifths of an inch). It has two swellings or enlargements, the cervical and lumbar. Their positions, size, and extent are shown on the diagram. Its specific gravity is 1.030.

It is surrounded by three membranes, all of which are continuous with the corresponding envelopes of the brain. They are the dura mater, the arachnoid, and the pia mater (Fig. 103).

The *dura mater* is the external covering. It is a long sac attached to the edge of the foramen magnum above and extending down until its walls fuse together at the level of the second sacral vertebra. Its cavity, therefore, is much longer than the spinal cord. It is attached to the bony canal at its lower end, and is held loosely by twenty-two lateral ligaments (*l. denticulata*) throughout its length.

The *arachnoid* is a thin, semitransparent membrane lying loosely over the cord and roots.

Internally this is connected by numerous connective-tissue filaments with the innermost membrane, the *pia mater*. The latter is a thin vascular sheath applied closely to the cord and roots. The space between the dura and arachnoid is called the arachnoid cavity. It contains a very little cerebro-spinal fluid. That between the arachnoid and pia is called the subarachnoid cavity; it contains a good deal of cerebro-spinal fluid. Both cavities connect with those of the brain and probably with each other. The dura has a mechanical protective function, the arachnoid a serous and the pia a vascular function.

The spinal cord is movable in its canal to the extent of from one-half to one inch.

The Nerve Roots.—The spinal nerve roots are covered with the pia and arachnoid. They pierce the dura in two places and unite to form a mixed nerve. The dura mater is prolonged over the nerves as they pass through it, forming a tubular sheath. The anterior roots are the larger. At the point of exit of the nerves from the cord a slight constriction is formed.

The Root Ganglia.—On each posterior root, *outside the dura*, is a posterior spinal ganglion. The ganglia lie in the intervertebral canals, except those on the sacral nerves.

Fissures.—Throughout the whole length of the cord there are two median

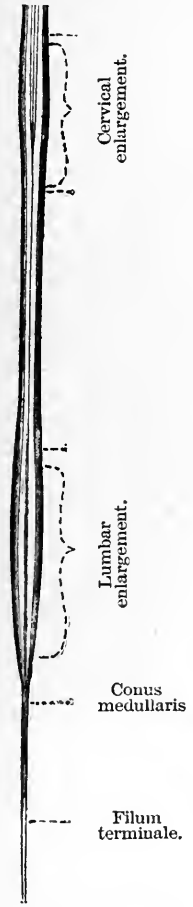


FIG. 102.—SHOWING THE RELATIVE SIZE OF THE DIFFERENT PARTS OF THE CORD (After Allen).

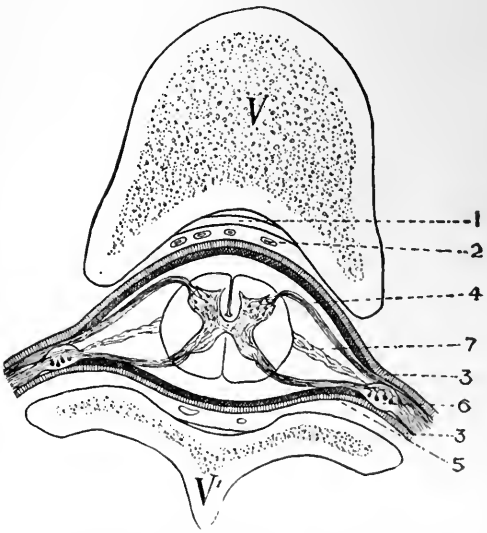


FIG. 103.—V, Spinous process; V', body of vertebra; 1, ligament; 2, vessels; 3, dura mater with the arachnoid lying directly beneath it; 4, anterior root; 5, posterior root; 6, spinal ganglion; 7, ligament.

fissures, called the anterior and posterior.

Columns.—These fissures and the lines formed by the exit of the roots divide the cord into four columns—anterior, posterior, and two lateral.

The Composition of the Cord.—The cord is composed of white and gray matter. The white matter lies outside and is composed

mainly of nerve fibres, the gray matter mainly of nerve cells. Each has also neuroglia, connective tissue, and blood-vessels. In the gray matter in a central canal lined with epithelial cells.

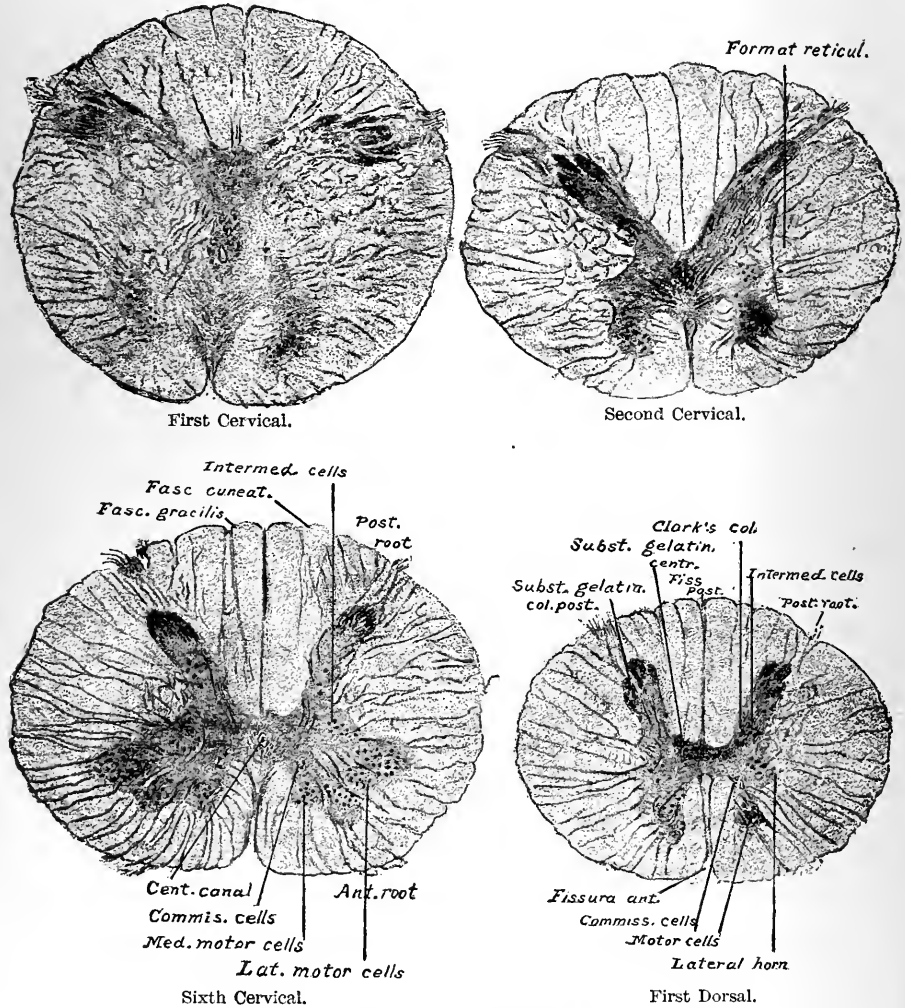


FIG. 104 *a*.—SHOWING ARRANGEMENT OF GRAY AND WHITE MATTER AT DIFFERENT LEVELS OF THE CORD (After Merkel).

The gray matter is arranged, as shown in the figures, somewhat in the shape of a letter H. Its different parts are called the anterior and posterior horns and intermediate gray. At certain levels there are lateral horns. The gray matter changes in shape at dif-

ferent levels of the cord. It is greatest in amount at the lumbo-sacral junction (23.33 sq mm.); next at the cervical enlargement (sixth cervical) (17.32 sq. mm.). It increases in amount relatively to the white matter from above downward (Fig. 104). The gray matter of the two halves of the cord is connected by a bridge or commissure. The anterior part is composed chiefly of white medullated nerves, and is called the white commissure. The posterior is composed of very fine nerve fibres, mostly medullated collaterals,

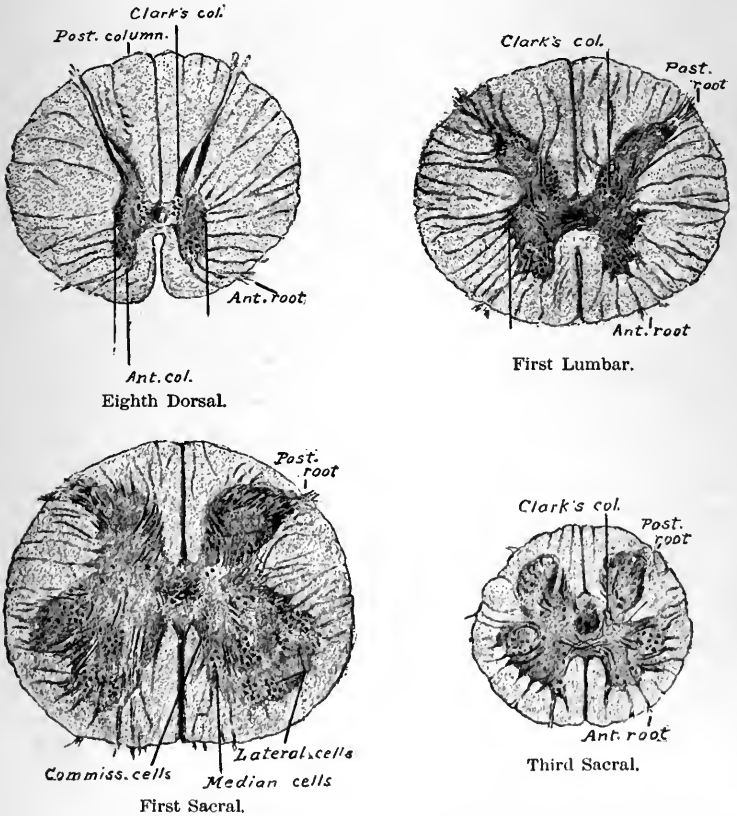


Fig. 104 *b*.—Showing Arrangement of Gray and White Matter at Different Levels of the Cord (After Merkel).

called the gray commissure. Between the two is the central canal, and surrounding it is the *central gelatinous substance*, composed of neuroglia.

The posterior horns reach to the periphery. They are divided, beginning from without, into the rim zone or Lissauer's column,

the spongy zone, and the gelatinous substance of Rolando. The rim zone is composed of very fine nerve fibres; the spongy zone and gelatinous substance are composed of very small nerve cells, some having continuous neuraxons (cells of Deiter) and some having rapidly branching neuraxons (cells of Golgi). The substance of Rolando is extremely rich in nerve cells and is not made up of neuroglia, as was once supposed.

To sum up, we have the gray matter—

Arranged in:	Composed of:
1. Horns { anterior, lateral, posterior.	*a*. A ground substance of neuroglia and connective tissue forming the substantia spongiosa.
2. Intermediate gray.	*b*. Cell groups: internal, anterior, etc.
	c. Plexuses of fine nerve fibres, *e.g.*, in the rim zone.
	d. Masses of neuroglia: (1) The central gelatinous substance. (2) The periphery of the cord and the spongy zone.
	e. Blood-vessels and connective tissue.

Now, taking up some of these factors in detail, we find that:

(*a*) The ground substance of the gray matter is made up of a fine meshwork of fibres which are the processes of neuroglia cells and of nerve cells. Besides this, there is some connective tissue, and there are prolongations from the base of the epithelial cells lining the central canal (Fig. 105).

(*b*) The nerve cells are, in part, arranged in groups with the long axis parallel to that of the cord. The cells are surrounded by a rich plexus of dendrites and end brushes, as well as by the supporting neuroglia matrix, a little connective tissue, and many small blood-vessels. The cell groups are named in accordance with their position—internal, antero-lateral, lateral, median, posterior or sensory cells, and the cells of Clark's column (Fig. 104).

This nomenclature answers for ordinary anatomical descriptions. Histologically we find two kinds of cells, the root cells and column cells (Strangzellen). The former are those cells whose neuraxons pass out to form the anterior roots. They form the great part of the anterior horns. Deep in the anterior horns are a few root cells, whose neuraxons pass into the posterior roots and thence to the ganglionic system. The column cells are found in the posterior horns, intermediate gray, and to some extent in the anterior horns. Their neuraxons pass to the white matter of the same or opposite side, and furnish commissural, associative, and even long column fibres.

The anterior-horn root cells are arranged in groups which overlap each other. Each group has the special duty of presiding over certain sets of muscles or other organs which have a common function. These cells are large in size, 35 to 100μ ($\frac{1}{700}$ to $\frac{1}{250}$ in.); they are multipolar, having five or six processes, one of which is an axis-cylinder process, which, in lower animals at least, gives off

a collateral before it leaves the cord. The cells in the central parts of the horn are the smaller; the cells in the lumbar swelling are largest, because they are connected with long nerves. The cells of the cervical swelling are next in size. The cells of the posterior horn are small and multipolar. The cells of Clark's column are bipolar, 30 to 60μ ($\frac{1}{800}$ to $\frac{1}{400}$ in.) in diameter, and are arranged

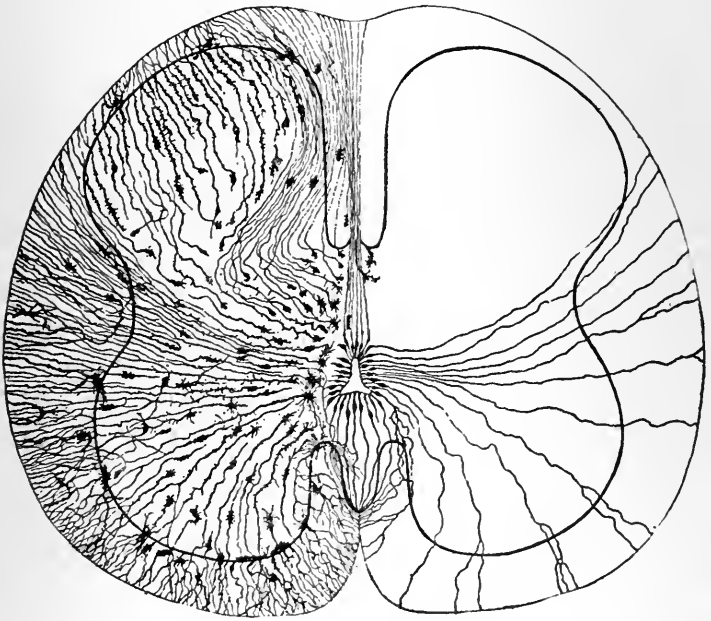


Fig. 105.—Showing the Neuroglia Groundwork in an Embryonic Cord.

with their long diameter parallel to the axis of the cord. They are grouped together in a kind of nest at the inner and central part of the posterior horn (see Fig. 104). Clark's column is most distinct from the eighth dorsal to the second lumbar nerves, but extends up as far as the last cervical. An analogous group of cells is found at the level of the second and third sacral nerves. A small group of spindle-shaped cells lies in the intermediate gray matter at the base of the posterior horns. There are other minor groups of cells which it is not necessary to describe here.

The white matter of the cord is composed mainly of neuraxons and the collaterals of these running in a supporting network of neuroglia, connective tissue, and blood-vessels. Surrounding it, and lying just beneath the pia mater, is a thin layer of neuroglia 5 to 50μ ($\frac{1}{5000}$ to $\frac{1}{500}$ in.) thick. The neuraxons are medullated, but have no neurilemma, and but few, if any, nodes of Ranvier. There are two kinds: the large (81 to 20μ) and the small (2 to 3μ in

diameter). The small fibres make up the postero-internal (Goll's) column entirely, and are numerous in the deep part of the lateral columns, but they are found in all regions. The fibres run up and down for the most part, but constantly send off branches to the gray matter. They are arranged in columns, the division being based partly on anatomical, partly on physiological, and partly on embryological grounds.

Anatomically there is a simple and natural division, which we have already given, into the anterior, lateral, and posterior columns, the divisions being made by the median fissures and the roots of the nerves.

On physiological and embryological grounds the columns are further subdivided as follows:

- The anterior columns are divided into
  - Direct pyramidal tract.
  - Anterior fundamental column.
- The lateral columns are divided into
  - Lateral fundamental columns.
  - Lateral limiting layers.
  - Crossed pyramidal tracts.
  - Direct cerebellar tracts.
  - Antero-lateral ascending and descending tracts, or Gowers' column.
- The posterior columns are divided into
  - Postero-internal column, or column of Goll.
  - Postero-external columns, or column of Burdach.
    - Burdach's column is divided into
    - Middle root zone.
    - Posterior root rone.
  - The ventral zone.
  - The comma.
  - The oval zone.
  - The triangular column.
  - Rim zone, or column of Lissauer

The fibres which make up these columns are of two kinds—*long* or projective, *short* or associative.

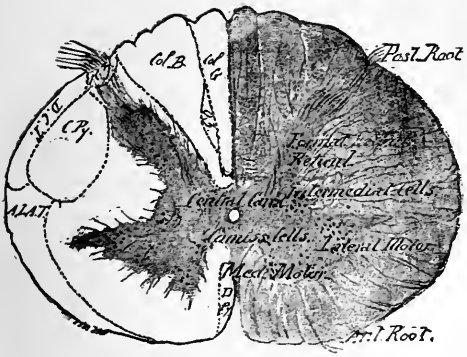
The *long* fibres connect the different levels of the cord with the brain, and the posterior spinal ganglia with nuclei in the upper part of the cord.

The *short* or associative fibres connect different levels of the cord with each other, and also connect the two halves of the cord at the same levels.

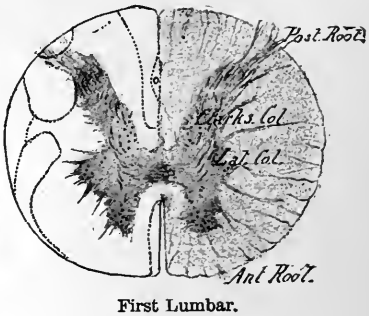
The names of the long-fibre tracts are the direct and crossed pyramidal, the direct cerebellar, the antero-lateral ascending, and the postero-internal or column of Goll.

The *direct pyramidal tract* lies along the anterior median fissure and extends down as far as the lower part of the dorsal cord. Its fibres cross over in the anterior commissure at various levels and connect with the cells of the anterior horns.

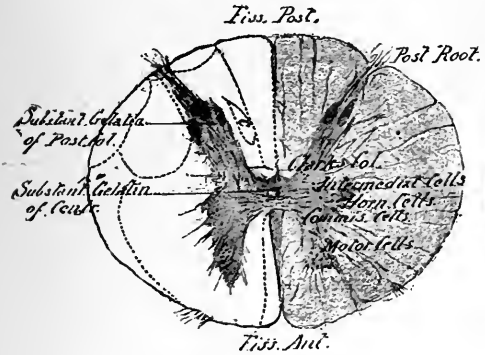
The *crossed pyramidal tract* extends down the whole length of the lateral column of the cord and sends its fibres to the anterior horns of the same side.



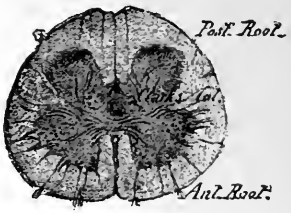
Sixth Cervical Segment.



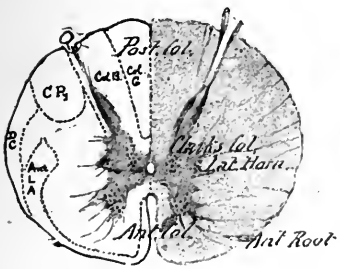
First Lumbar.



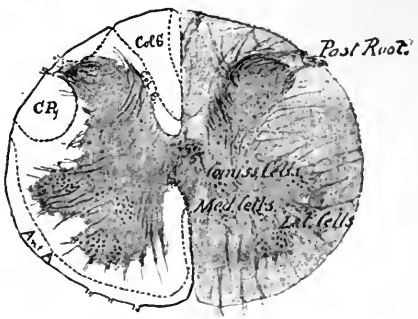
First Dorsal.



Fourth Sacral.



Eighth Dorsal.



First Sacral.

FIG. 106.—SHOWING THE ARRANGEMENT OF THE GRAY AND THE WHITE MATTER AT DIFFERENT LEVELS OF THE CORD, also the columns and cell groups.

Both of the above tracts are continuations of the anterior pyramids or motor tracts of the medulla. These pyramids divide at the lower end of the medulla, about ninety per cent of fibres crossing over to form the crossed pyramidal tract and ten per cent continuing on the same side. Some of the fibres of the crossed pyramid redecussate (in lower animals) and enter the pyramidal tract of the side on which they started.

The *direct cerebellar tract* begins at the level of the first lumbar nerves. Its fibres originate in the vesicular column of Clark. They pass up to the cerebellum and go chiefly to the vermis. Most of them then cross over and enter the red nucleus.

The *antero-lateral ascending column* extends nearly the whole length of the cord. Its fibres come from the anterior commissure and the sensory cells of the opposite posterior cornu. They pass up and end in the lateral nucleus.

The *postero-internal column*, or column of Goll, is composed of fibres which originate in the ganglia of the posterior roots, pass inward, and without crossing ascend, to end in a nucleus at the upper limit of the cord, the nucleus of Goll's column (postero-internal nucleus). The column extends the whole length of the cord. It is very small in the sacral region, but increases in size as it passes up.

There are a few long fibres scattered in the anterior and lateral ground bundles. They degenerate down and are called the *antero-lateral descending tract.*

The names of the short-fibre columns are the anterior and the lateral fundamental columns, the lateral limiting layer, and the column of Burdach. This latter column contains in its cervical part some long fibres which end in a nucleus at the upper limit of the cord, the postero-external nucleus of the column of Burdach.

The posterior columns also contain three short-fibre columns whose cells of origin lie in the gray matter of the cord. The fibres in the cervical region lie in the shape of a comma (comma of Schultze), in the lumbar region in the shape of an oval (oval zone of Flechsig), in the sacral zone in the shape of a triangle (triangle of Gombault). Besides this there is a zone of short fibres lying close to the gray commissure the whole length of the cord—ventral zone or posterior fundamental column.

The Relations of the Different Parts of the Spinal Cord to the Peripheral Nerves, to the Brain, and with Each Other.—I will begin with a description of the way in which the anterior and posterior nerve roots are connected to the cord; then describe the mode in which the different columns and cell groups are connected with each other; and finally I will indicate briefly the connections of the cord with the brain.

The *anterior nerve roots* are connected directly with the anterior-horn cells, of which they are processes, and together with which they form the peripheral motor neurons. It is possible that in man they send off collaterals before leaving the cord.

The cells of the anterior horn are surrounded by two chief sets of "end brushes," one coming from the pyramidal tracts, the other from the posterior horns and roots. Thus these cells are in relation with impulses from the brain and from the periphery.

The *posterior nerve roots* originate in the spinal ganglia. On entering the cord the fibres divide like a T and pass up for one or more inches, and down for a short distance only. They then enter (1) the column of Goll, (2) the anterior and posterior commissure, or (3) the posterior horn. They all send off collaterals, and termi-

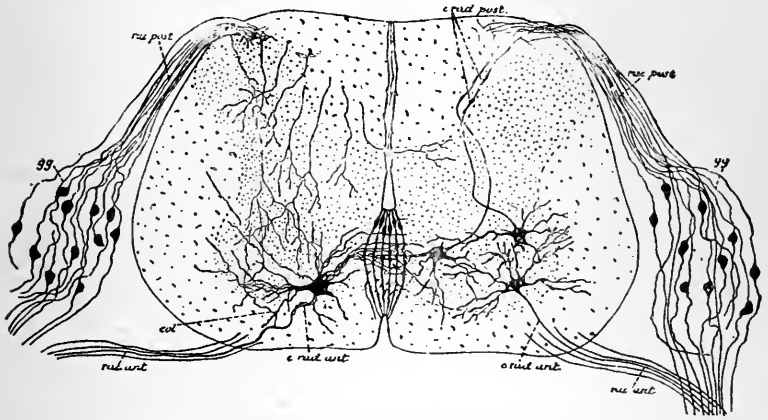


FIG. 107.—SHOWING THE CELLS OF ORIGIN OF THE MOTOR NERVES IN THE ANTERIOR HORNS OF THE SPINAL CORD, AND THE CELLS OF ORIGIN OF THE SENSORY NERVES IN THE POSTERIOR SPINAL GANGLIA (Van Gehuchten).

nate eventually in end brushes surrounding nerve cells, which form their terminal nuclei. These root fibres, with their cells of origin and the sensory nerve fibres, form the peripheral sensory neurons.

There are at least three groups of nerve fibres which enter by the posterior roots and make different connections with the cell groups or columns:

1. An innermost set. These pass across the postero-external column and enter the median or Goll's column, which they ascend, to end in the nucleus.

2. A median set. These pass along the inner side of the posterior horn, and end either (*a*) in cells of the deeper part, (*b*) in the spindle-shaped cells, or (*c*) go to the anterior horn; still others (*d*) cross over in the commissure to enter the antero-lateral tract.

3. An outer set. These are very fine fibres which enter the tip or outer part of the posterior horn, and then run up and down, forming the rim zone. They eventually connect in the usual way with the sensory cells of the posterior horn.

The different parts of the spinal cord are connected by the short fibres which unite different levels of the cord, and by commissural fibres which unite the different halves of the cord. These short

and commissural fibres originate in groups of nerve cells lying in the central parts of the gray matter and are called associative or column nerve cells. They are small and multipolar. Some are distributed sparsely in the white columns. Fibres arise from them, run in the commissures and short-fibre tracts, and end in brushes which put the fibres in relation with various cell groups (Figs. 109, 110).

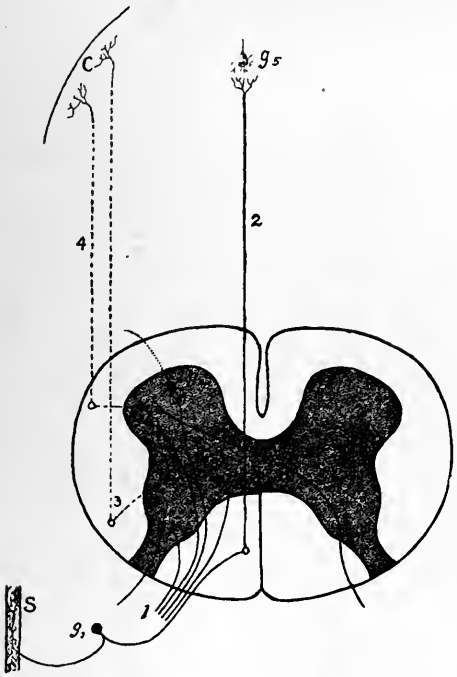


FIG. 108.—SCHEMATIC DRAWING OF SENSORY TRACTS. g_1, Spinal ganglion cell with one neuraxon going in the peripheral nerves to the skin *s*, and with one in the posterior root; 1, fibres of the posterior root, ascending partly in the posterior column (2), partly entering the posterior horn; g_2, cell of Clarke's column, passing from this to the cerebellar tract (3); g_3, ganglion cell in gray matter, sending fibres (4) to Gowers' tract; g_5, cell in nucleus gracilis in medulla oblongata; from posterior root fibres also pass to g_4, anterior horn ganglion cell.

PHYSIOLOGY.—The detailed facts regarding the functions of the spinal cord may be gotten in physiological text-books. I shall give only those bearing more or less directly on the localization of the functions. The spinal cord is a conductor and centre of nervous action. It represents the lowest evolutionary level of the development of the nervous system. Its functions, so far as they are independent, are stable and well organized, but of a mechanical and relatively simple order.

Functions of the White Columns.—The white matter is a conductor of nerve impulses, and its functions are relatively simple. We have only to study the direction and kind of impulses carried by its various columns. The *direct* and *crossed pyramidal tracts* originate from cells in the motor cortex of the brain. They carry motor impulses downward from the brain. The crossed pyramidal tract crosses in the medulla to the side opposite to that where it originates, and passes down the lateral column to connect the motor cells of the anterior horns. The direct pyramidal tract runs along in the anterior column, and at different levels sends fibres across through the anterior commissure to the motor cells of the anterior horns. These tracts normally exercise a continual inhibitory in-

fluence on the motor cells of the anterior horns, so that when destroyed there develop spasmodic conditions of the paralyzed part.

The anterior ground bundle, lateral ground bundle, and *lateral limiting layer* have the function of associating different levels of the cord and of connecting it also with nuclei in the medulla and centres in the cerebellum.

The *columns of Goll* conduct special sensations from the muscles, articulations, and tendonous sheaths *via* the root on the same side. When diseased, there is a loss of the sense of position of the limbs, of the power of estimating weights, and of co-ordination of muscular effort (ataxia). The fibres cross over in the medulla.

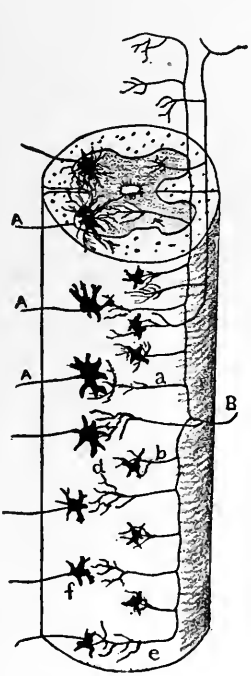


Fig. 109.

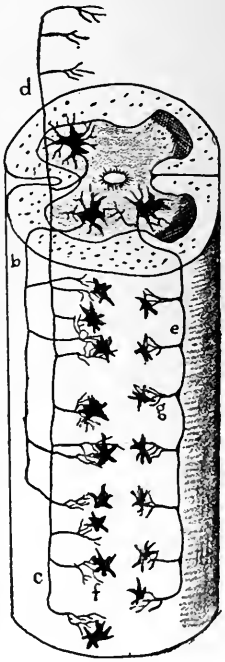


Fig. 110.

Fig. 109.—Showing the Connections of the Anterior and Posterior Roots and Cornua with Each Other (Cajal). *A*, Anterior root; *B*, posterior root; *a*, collaterals; *d*, end-brushes.

Fig. 110.—Showing the Association or Short-Fibre System of the Cord (Cajal). *a*, Anterior cornua cell; *b*, *c*, *d*, association fibres; *e*, posterior association fibres.

The *columns of Burdach* conduct to a certain extent tactile sensations coming in from the opposite posterior root. They also contain many associative fibres, connecting different levels. Pain-sense fibres and excito-reflex fibres from the posterior roots run

through these columns to reach the commissure or the anterior horns; other fibres run through it to Clark's column and to the column of Goll. Hence it is a pathway for all kinds of afferent impulses. When diseased, there may be pain, anæsthesia, ataxia, and loss of reflexes. The fibres cross over at once to the opposite side. The direct cerebellar tracts carry impulses to the cerebellum which assist in maintaining equilibrium.

The *antero-lateral ascending tract* conducts sensations of pain and temperature, coming in from the opposite side, through the anterior commissure.

There are considerable variations in the paths of conduction of tactile temperature and pain sensations, and their exact position is not positively known. In transverse lesions of the cord these tracts do not degenerate upward so completely or uniformly as do secondary degenerations of other long-fibre tracts. Hence they probably receive some interruptions in their course. In fact, it is probable that the paths for these impulses are very wide and not arranged in a compact bundle like the motor tract.

The *gray matter* contains chiefly cell groups which act as centres and distributors of nerve impulses. In the *anterior horns* the cells have a motor and trophic function. The larger cells are at the outer parts of the horn and send fibres to the larger skeletal muscles. The more central cells are connected with small muscles and those having more delicate functions and adjustments. In the still more central and intermediate part, also, are separate trophic cells for the muscles, bones, and joints, and cell groups which preside over vasomotor and secretory functions. Among these groups are the *spindle-shaped cells*, which send fibres to the vasoconstrictors (Gaskill), through the anterior (Hill) and perhaps posterior roots (Gaskill). The cells of Clark's column receive fibres from the viscera. Impulses pass to these cells and thence to the direct cerebellar tract and cerebellum. Their function is to conduct impulses from the viscera relating to equilibrium and sense of position. They are analogous to the fibres of the column of Goll. According to Gaskill, Clark's column is a centre for the vasodilators. This is unlikely.

Automatic Centres.—The nerves and cells of the cord are arranged in complex groups which preside over certain functions or respond in a definite way to certain stimuli. These are called the spinal automatic centres. They are the cilio-spinal, secretory, vasomotor, genital, vesical, and rectal. The important parts of these centres lie deep in the gray matter on either side of the central canal, but nearer the base of the posterior horns. Lesions of the white matter, or of the anterior or posterior horns, do not directly affect them.

The *cilio-spinal centre* reaches from the seventh cervical to the second dorsal segment, inclusive. Its stimulation causes the pupils to contract.

The *genital centres*, including those for erection and ejaculation, reach from the first to the third sacral segment, inclusive.

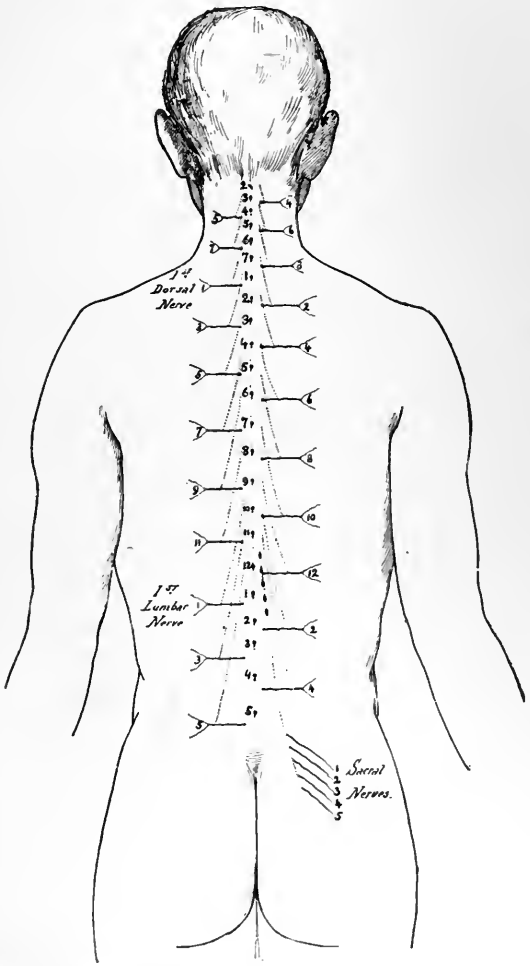


FIG. 111.—SHOWING THE RELATION OF THE SPINOUS PROCESSES TO THE POINTS OF ORIGIN IN THE CORD OF THE SPINAL NERVES.

The *bladder* and *rectal centres* are in the fourth and fifth sacral segments, extending up and down a short distance, the bladder being perhaps a little lower.

The spinal *vasomotor centres* extend from the second dorsal to

the second lumbar segments. The vasodilator nerves pass out by the anterior, the constrictor by the posterior root (Gaskill).

The *cells of the posterior horns* are sensory in function and are connected with the tactile, pain, temperature, and reflex fibres of the posterior roots.

The *trophic centres for the joints, bones, and skin* apparently lie near the posterior horns. Their fibres pass out by the posterior roots.

Topography and Localization.—The neurologist and surgeon need to know, for purposes of diagnosis:

1. The relation of the spinal nerve roots, at their point of origin, to the spinous processes. This is shown in the figure (p. 221). In general it will be seen that the different pairs of nerve roots arise opposite the spinous process of a vertebra one or two segments above those between which it makes its exit. Thus the sixth cervical originates opposite the fourth cervical spine, the sixth dorsal between the third and fourth dorsal spines, the first lumbar between the eleventh and twelfth dorsal spines. There is considerable variation in these relations.

2. The next points desired are the special function of each pair of nerve roots anterior and posterior, and the level of the various centres in the cord. This is shown in the following table, based on that originally devised by Starr, modified by Mills, Sachs, and myself from personal experiments and the clinical and pathological observations of Thorburn and others.

Muscles of Tongue, Palate, and Pharynx.

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Genio-glossus.	Pushes tongue to opposite side.	Tongue when protruded deviates to paralyzed side.	The twelfth nerve (hypoglossal).	Medulla.	Bulbar palsies (acute and chronic); in specific and tubercuous diseases of base; dystrophies (rare).
Styloglossus.	Raises tongue backward and upward.	Tongue cannot be moved backward or hollowed out (action deficient in many healthy subjects).	The twelfth nerve.	Medulla.	
Lingual muscle proper.	All movements of the tongue itself.	When lying in mouth deviation to healthy side; when protruded deviates to paralyzed side; if one or both halves are atrophied tongue looks shrivelled.	The twelfth nerve.	Medulla.	
Azygos uvulæ.	Shortening of uvula.	Uvula deviates toward sound side; if both sides are paralyzed there are nasal tone and regurgitation through nose.	Probably pharyngeal plexus; seventh nerve (?).	Medulla.	As above.

MUSCLES OF TONGUE, PALATE, AND PHARYNX.—*Continued.*

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Levator palati.	Raises the velum palati.	Arch cannot be raised in the intonation of "ah;" if paralysis is bilateral flapping of arch and regurgitation of food through nose.	As above.	Medulla.	As above; see also seventh-nerve affections.
Palato-pharyngeal muscles.	Prevent food from passing toward upper part of pharynx and posterior nares.	Regurgitation of food; nasal speech.	The fifth nerve.	Pons.	Basilar affections.
Stylo-pharyngeus.	Helps to draw larynx upward so as to be closed by epiglottis and overtopped by tongue.	Imperfect deglutition; food gets into windpipe.	Glosso-pharyngeal.	Medulla.	Bulbar affections and diseases of the base.
Constrictors of pharynx.	Help to push food into gullet.	Food is swallowed very imperfectly; sticks in throat.	Pharyngeal plexus.	Medulla.....	Diseases of the base (bulbar).
Laryngeal muscles.	Movements of vocal cords in respiration and in articulation.	Hoarseness and difficulty in breathing; laryngoscopic examination reveals false position of vocal cords (see special text-books).	Recurrent laryngeal nerve excepting the crico-thyroid muscle.	Medulla.....	Bulbar troubles (similar symptoms may be caused by tumors and foreign bodies in larynx).

MUSCLES OF HEAD AND NECK.

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Sterno-cleido-mastoid.	Raises and turns face to opposite side; head inclines to same side; if both muscles act conjointly head is brought forward.	Inability to raise head from bed, or other horizontal position, if both muscles are affected; if one muscle is affected, no marked change of position, unless opposite muscle is contractured; spasm of muscle frequent; head inclined to one side.	Spinal accessory.	Medulla and second and third cervical segments.	In bulbar and cervical-cord affections; in later stages of progressive muscular atrophies; occasionally in neuritis.

MUSCLES OF HEAD AND NECK.—*Continued.*

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Rectus capitis anticus major.	To flex head.	Cannot flex head so as to bring chin on chest.	Upper cervical.	Upper cervical segments.	Diseases of the cervical region (myelitis, meningitis, tumor; progressive wasting of muscles).
Rectus capitis anticus minor.	To flex head.				
Rectus capitis lateralis.	Slight rotation.	Deficient rotation scarcely noticeable, unless sterno-cleido-mastoids are diseased.			
Scaleni anterior medius, et posterior.	Elevate ribs when vertebral column is fixed; aid in inspiration; slight lateral flexion.	Deficient inspiratory movements.	Lower cervical nerves.	Upper cervical segments.	
Longus colli.	Flexion of vertebral column.	Imperfect flexion of upper spine.	Lower cervical nerves.		

MUSCLES OF SHOULDERS AND UPPER EXTREMITY.

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Trapezius. 1. Clavicular portion (respiratory; outer third of clavicle to occipital bone).	Pulls head backward; rotates slightly toward side of muscle, so that chin is turned to opposite side; contraction of both clavicular portions bends head backward; slight elevation of shoulders; aids in deep inspiration.	Deficient backward movement of head; not marked as a rule because deep muscles perform this function; shoulder does not move during inspiration.	Spinal accessory.	Medulla and second and third cervical segments.	Progressive muscular wasting; diseases of medulla and upper cervical cord; clavicular portion least frequently involved.
2. Middle portion (from acromion and outer spine of scapula to ligament. nuchæ and upper dorsal spines).	Raises shoulder-blade; elevation of acromion (clavicle goes along).	Acromion depressed by weight of upper extremity; inner upper angle may be pulled upward by levator anguli scapulæ; internal lower angle is nearer to median line.	Spinal accessory nerve.	As above.	As above.

MUSCLES OF SHOULDERS AND UPPER EXTREMITY.—*Continued.*

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
3. Lower portion and adductor.	Adduction of scapula toward median line.	Margin of scapula is about ten cm. distant, instead of being five or six cm. distant from median line; loss of adductor may be covered up by action of rhomboids; rounding of back.	Spinal accessory nerve.	Medulla and second and third cervical segments.	
Rhomboids.	Oblique movement of scapula from below, upward and inward, so that inferior angle is brought nearer the median line; holl spinal margin of scapula down to thorax.	Deep groove between inner margin of scapula and thorax; if serratus is normal, this groove disappears if arm is extended forward; shoulder blade cannot be approximated to median line. (According to Duchenne this can be effected by upper portion of latissimus dorsi.)	Fifth cervical.	Fourth and fifth cervical segments.	As above.
Levator anguli scapulæ.	Draws superior inner angle of scapula upward; aids in shrugging of shoulders.	Isolated paralysis rare.	Third and fifth cervical nerves.	Second and fourth (?) cervical segments.	Dystrophies and cervical diseases.
Serratus magnus.	Rotation of shoulder-blade outward, and slight elevation of acromion; holds inner margin of scapula to thorax; brings arm from horizontal to vertical position.	Scapula pulled upward; lower inner angle nearer the median line; arm cannot be raised above horizontal position; if arm is stretched forward scapula is removed from thorax ("winged scapula"); during abduction of arm, scapula is moved nearer to median line, and crowds trapezius and rhomboids forward.	Posterior thoracic nerve.	Fifth and sixth cervical segments.	Progressive muscular atrophies (dystrophies); neuritis of part of the brachial plexus; after traumatic injuries to shoulder: in cervical-cord affections.
Deltoid (three divisions).	To raise arm to horizontal position, and forward, outward, or backward; movements possible only if scapula is fixed by action of serratus and trapezius.	Can raise shoulder but not arm; shoulder flattened (atrophy); groove between acromion and head of humerus; each division of deltoid may be paralyzed singly.	Circumflex.	Fourth, fifth, and sixth cervical segments.	As above; also in Erb's form of obstetrical paralysis.
Infraspinatus. Teres minor.	Rotator humeri posticus (Duchenne); rotate arm outward.	Arm cannot be moved outward. Difficulty in writing (Duchenne).	Suprascapular. Circumflex.	Fourth, fifth, and sixth cervical segments.	As in case of deltoid.
Subscapularis.	Rotator humeri anticus (Duchenne); rotates arm inward.	Arm cannot be moved inward; scapula is rubbed against ribs.	Subscapular nerve.		

MUSCLES OF SHOULDERS AND UPPER EXTREMITY.—*Continued.*

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Supraspinatus.	Helps to steady shoulder-joint and to elevate arm forward and outward; outer angle of scapula is depressed.	According to Duchenne, humerus is separated still farther from acromion, if supraspinatus is affected in addition to deltoid.	Suprascapular.	Fourth cervical.	As above.
Latissimus dorsi.	Pulls the arm when raised, downward and backward; if arm is at rest upper portion brings scapula nearer the median line; united action of upper third of both muscles causes extension of dorsal trunk; single action causes lateral movement of trunk.	Arm cannot be moved backward; insufficient extension of dorsal spine; trunk cannot be moved laterally.	Subscapular, also branches of dorsal and lumbar nerves passing through muscle.	Sixth and seventh cervical.	As in progressive atrophies and dystrophies; in cervico-dorsal lesions; in neuritis.
Teres major.	Rotates raised humerus inward; adduction of arm to thorax; slight elevation of shoulder.	Very few symptoms; action supplied by other muscles.	Subscapular.	Seventh cervical.	As above.
Pectoralis major.	Clavicular portion depresses humerus from raised position to horizontal; adduction of arm, as in giving a blessing; sternal portion depresses arm completely, and if arm is at rest draws acromion forward and backward.	Imperfect adduction of arm; paralysis can be discovered best by extending arms and trying to press volar surfaces against each other.	Anterior thoracic.	Fifth, sixth, and seventh cervical.	Amyotrophies and dystrophies, chiefly; also in lesions of brachial plexus.

Muscles of Arm, Forearm, and Hand.

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Triceps....	Extends forearm; long head of triceps, and coraco-brachialis help to keep head of humerus in position.	Arm cannot be extended except by its own weight; if long head of triceps is affected subluxation of head of humerus occurs easily.	Musculo-spiral.	Sixth, seventh, eighth cervical segments.	Poliomyelitis and other affections of cervical cord; traumatic injuries; amyotrophies and dystrophies (triceps escapes in many peripheral palsies.)
Biceps.....	Flexion and supination of forearm.	Flexion deficient, but can be carried out in part by other muscles.	Musculo-cutaneous.	Fourth, fifth, sixth cervical.	
Supinator longus.	Flexes forearm and aids in pronation.	Flexion and pronation deficient; muscle does not stand out prominently if arm is flexed and attempt is made by another to extend it forcibly; if muscle is atrophied arm is spindle-shaped.	Musculo-spiral.	Fourth, fifth cervical.	As above; involved in peripheral neuritis (traumatic), not in lead palsy.
Supinator brevis.	Supinates hand when forearm is extended.	Deficient supination of hand.	Musculo-spiral.	Fifth cervical.	Diseases as above; also in peripheral palsies.
Extensor carpi radialis longus et brevis.	Extension and abduction of wrist; the shorter muscle has pure extension action only.	Wrist cannot be flexed dorsally (extended) or abducted; flattening of forearm.	Musculo-spiral.	Seventh cervical.	As before; especially in neuritis.
Extensor carpi ulnaris.	Extension and abduction of wrist.	Wrist cannot be flexed dorsally or adducted; "drop-wrist" is characteristic of paralysis of extensors.	As above.	Seventh cervical.	As above.
Extensor digitorum communis.	Extension of first phalanges of all fingers and abduction.	First phalanges cannot be extended nor fingers abducted; grasp is weak because flexor muscles are shortened and cannot contract forcibly.	Musculo-spiral.	Seventh cervical.	As above.
Extensor indicis.					
Extensor minimi digiti.					
Flexor carpi radialis.	Flexion of wrist and pronation.	Deficient flexion.	Median.....	Eighth cervical.	As above.
Flexor carpi ulnaris.	Flexion of wrist and supination.	Flexion and supination impaired.	Ulnar.....	Eighth cervical.	As above.
Palmaris longus.	Flexion of wrist only.	Flexion impaired; no anomalous position of hand from paralysis of wrist as hand falls by its own weight; the flexors of fingers may act as substitutes.	Median	Eighth cervical.	As above.

MUSCLES OF ARM, FOREARM, AND HAND.—*Continued.*

Name of Muscle.	Normal Function.	Symptoms of Deficient Action.	Innervated by	Represented in	Diseases in which Muscle is commonly Involved.
Flexor digitorum sublimis.	Flexes second phalanx toward first.	Second phalanx cannot be flexed.	Median.....	Eighth cervical.	As above.
Flexor digitorum profundus.	Flexes last two phalanges toward first.	Last two phalanges cannot be flexed.	Ulnar and Median.	Eighth cervical.	As above; muscle should be tested with special care in cases of traumatic injuries.
Interossei and lumbricales.	Abduction and adduction of fingers if first phalanges are extended; flexion of first phalanges and simultaneous extension of second and third phalanges.	Fingers cannot be abducted or adducted; interosseous spaces are very marked; "Main en griffe" due to extension of first phalanges and flexion of second and third phalanges.	Ulnar, which also supplies third and fourth lumbricales; median supplies first two and sometimes third lumbricales.	Eighth cervical, first dorsal.	As above; often the first muscles to be affected in progressive spinal atrophies.
Thenar muscles: Extensor pollicis brevis.	Extends first phalanx and abducts metacarpal bone; acts with adductor pollicis longus.	Impairment of extension and adduction; flattening of ball of thumb.	Musculospiral.	First dorsal.	As before; more especially in amyotrophies and neuritis.
Extensor pollicis longus.	Extends both phalanges of thumb; also adduction of metacarpal bone and backward movement of thumb.	Deficient extension and adduction; second phalanx is flexed toward first.	Musculospiral.	First dorsal.	As above.
Abductor pollicis longus.	Abduction of metacarpal bone; aids in flexion of hand.	Deficient abduction of metacarpal bone; if this muscle and extensor pollicis brevis are paralyzed adduction results.	Musculospiral.	First dorsal.	As above.
Abductor pollicis brevis.	Opposition of thumb.	No opposition movement.	Musculospiral.	First dorsal.	As above.
Opponens pollicis and outer portion of the flexor brevis.			Median.		
Abductor pollicis brevis; flexor brevis and adductor.	Flex first phalanx and extend second phalanx (like interossei), also have an abduction and adduction action.	No flexion; if muscles are paralyzed and atrophied, ape hand is formed.	Median and ulnar.		As above.
Flexor pollicis longus.	Flexes end phalanx.	No flexion of end phalanx.	Median.		As above.

MUSCLES OF BACK AND LOWER EXTREMITIES.

Name of Muscle.	Innervated by	Symptoms of Deficient Action.
Erector spinæ; sacro-lumbalis; longissimus dorsi.	Dorsal nerves. Second to twelfth dorsal segments.	Lordosis of lower spine; perpendicular line from shoulder falls behind os sacrum; unilateral palsy causes deflection of spine toward sound side.
Abdominal muscles.	Dorsal nerves. Second to twelfth dorsal.	Lordosis with protrusion of nates and abdomen; other actions deficient; cannot straighten up from recumbent position without assistance of hands.
Quadratus lumborum.	Lumbar nerves.	Lateral movements of lower vertebræ imperfect.
Adductor muscles.	Obturator nerve, great sciatic and crural.	No adduction; thigh rolls outward.
Sartorius.	Crural. Third lumbar segment.	Flexion impaired; acts imperfectly.
Quadriceps femoris.	Crural. Third lumbar.	Leg cannot be extended; to test it ask patient, who is lying down with hip bent, to stretch out the leg; when patient is sitting down to extend leg.
Ilio-psoas.	Crural (lumbar plexus). Fourth lumbar.	Flexion difficult; in bed thigh cannot be flexed; difficulty rising from horizontal position.
Tensor fasciæ latæ.	Superior gluteal. Fourth lumbar.	
External rotators: Pyriformis. Gemelli. Quadratus femoris.	Sacral plexus (muscular branches). Fifth lumbar.	Deficient outward rotation; leg turned inward.
Internal obturator. External obturator.	Obturator nerve (lumbar plexus).	
Gluteal muscles.	Inferior gluteal (sacral plexus). First and second sacral. Gluteal superior. First and second sacral.	No extension of thigh; great difficulty in climbing; no abduction of thigh; waddling gait, exaggerated movement of pelvis.
Biceps; semitendinosus and semimembranosus.	Sciatic. Fifth lumbar segment.	Deficient flexion; action of quadriceps may cause excessive extension; in standing thigh is flexed to excess; trunk moved backward.
Gastrocnemius (also plantarius and soleus).	Internal popliteal. Fifth lumbar.	Deficient flexion of foot; heel cannot be raised; cannot stand on tiptoes.
Anterior tibial muscles (tibialis anticus, extensor digitorum, and extensor pollicis longus).	Anterior tibial. Fifth lumbar and first sacral.	Deficient extension; "dropfoot," toes scrape floor; to clear this, excessive flexion at knee and hip; contracture of flexors and pes equinus or equinovarus.
Peroneus longus.	Peroneal. First and second sacral segments.	Deficient abduction; plantar arch lessened; increased by contracture. Flatfoot; walking tiresome.
Posterior tibial muscle.	Posterior tibial nerve. First and second segments.	Deficient abduction or adduction; deformities result from deficiencies.
Peroneus brevis.	Peroneal. First and second segments.	
Interossei pedis et lumbricales.	Posterior tibial. First and second segments.	Abduction and adduction of toes deficient; paralysis of interossei; hyperextension of first phalanges; second and third flexed (clawed foot).
Adductor; flexor brevis and abductor hallucis.	Posterior tibial. First and second segments.	Deficient flexion of toes; foot cannot be pushed off ground easily.

THE BLOOD SUPPLY OF THE SPINAL CORD is a subject of great practical importance; and, as our knowledge of it has lately been increased, I shall present the matter here in some detail.

The spinal cord is supplied with blood by branches from the vertebral, ascending cervical, and superior intercostal arteries above, and by the dorsal intercostal, lumbar, and sacral arteries below. These send off small branches which enter the spinal canal

through the foramen magnum above and the intervertebral foramina at the sides; they pierce the dura mater and are distributed on the pia mater and in the cord. The arteries that thus supply the cord are these:

Primary Arteries.	Origin from	Ending in
Anterior spinal.	Vertebral (from subclav.).	Anterior median spinal artery.
Posterior spinal.	Vertebral.	
Lateral spinal.	Vertebral.	
Lateral spinal.	Ascending cervical (from subclav.).	Anterior and posterior spinal root arteries
Lateral spinal.	Superior intercostal (from subclav.).	Anterior and posterior spinal root arteries.
Lateral spinal.	Thoracic intercostal (from aorta).	Anterior and posterior spinal root arteries.
Lateral spinal.	Lumbar (aorta). Lateral sacral (from int. iliac).	Anterior and posterior spinal root arteries.

The anterior spinal arteries are branches of the vertebrals. They unite to form the anterior median artery, which runs down the whole length of the cord, receiving re-enforcements from the lateral arteries (Fig. 112). The *anterior spinal* arteries themselves nourish only a few upper segments of the cord. The anterior median artery is not, as has been taught, a true prolongation of the anterior spinals, but is really made up by the lateral spinals. In other words, the vertebral artery through its branches nourishes only the upper cervical region of the cord. The *posterior spinal* arteries are smaller than the anterior and unite on the posterior surface of the cord. They do not continue down as a posterior median artery—there is no such artery; but they help to form two plexuses on the postero-lateral surfaces of the cord.

The *lateral spinal* arteries are derived from branches of the subclavian artery as far down as the second dorsal root; below this point by the thoracic and abdominal aorta and the internal iliac. It is an interesting fact that at or a little below the point where the blood supply changes from the subclavian above the heart to the aorta below, pathological disturbances frequently occur (transverse myelitis).

Root Arteries.—The lateral spinal arteries, after they enter the spinal canal, are called the root arteries. They pierce the dura mater and pass, some along the posterior and some along the anterior roots, to the cord. There are about eight anterior-root arteries (five to ten) and about sixteen posterior-root arteries (see Figs. 112, 113). The anterior arteries are twice as large (one millimetre in diameter) and one-half as numerous as the posterior. The root arteries of the cervical region are rather the more numerous. There is a large and constant anterior-root artery in the dorso-lumbar region. The last two lumbar, the five sacral nerves, and the unpaired coccygeal nerve when it exists, are accompanied by small root arteries which do not reach up to the cord itself. The lower part of the spinal cord is supplied by large root arteries from

the lateral spinal arteries. Hence the theory of Moxon that the circulation here is feeble is not supported by Kadyi's investigations.

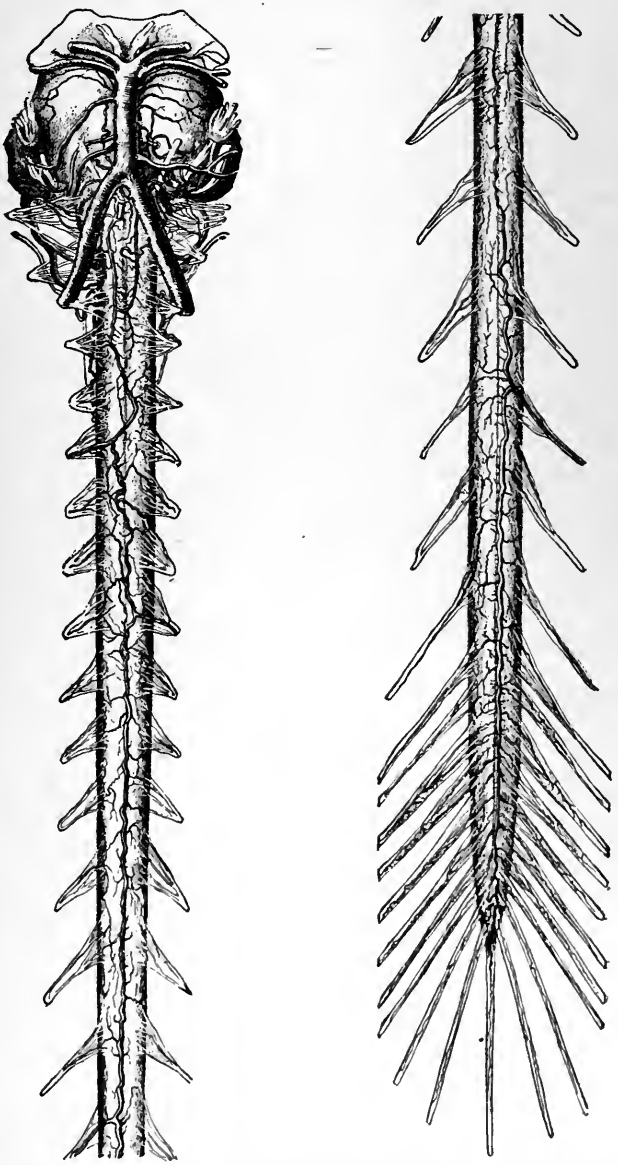


FIG. 112.—THE SPINAL CORD, ANTERIOR SURFACE, showing the nerve roots, root arteries, and anterior plexus (Kadyi).

The Plexuses.—The anterior root arteries pass to the anterior median fissure, and then divide, partly to form the anterior median artery and partly to form a rich plexus between the anterior roots; this is called the *anterior arterial plexus.* The posterior root arteries subdivide before they reach the cord, and send twigs to its lateral and posterior surfaces which form the *postero-lateral arterial plexus.* The posterior-root arteries do not anastomose to any extent with each other or form a posterior spinal artery, as is done by the anterior-root arteries. There are therefore three relatively independent arterial plexuses: the anterior plexus, the two postero-lateral plexuses.

Veins.—The veins of the spinal canal outside the dura mater have valves, those within it have none. The veins reach the pia mater and cord by passing along the nerve roots. Hence we have anterior and posterior *root veins,* corresponding to the root arteries, but more numerous, there being a total of forty or fifty. The an-

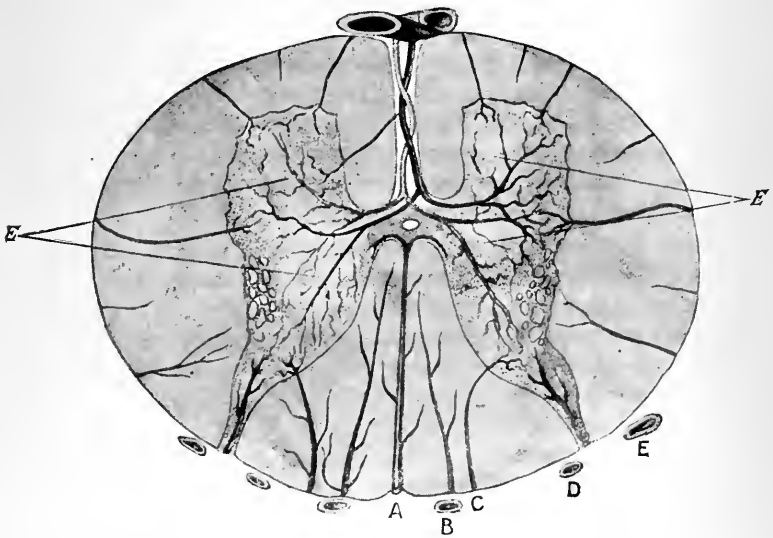


FIG. 113.—THE ARTERIAL SUPPLY. *A*, The artery of the posterior fissure; *B*, the interfunicular; *C*, artery of posterior horn; *D*, of posterior root; *E*, of posterolateral column.

terior-root veins are more numerous than the posterior, but smaller (twenty-five to twenty). The veins are a little larger than the arteries, the anterior veins being one-half to one millimetre, the posterior one and one-half to two millimetres, in diameter.

Thus we see that the posterior surface of the cord has more and smaller arteries, fewer but larger veins. The posterior surface is on the whole more richly supplied with veins, the anterior surface with arteries. The lateral surfaces are the least vascular.

	Number.	Size.
Anterior root arteries	5 to 10	1 mm.
Anterior root veins	25 to 30	½ to 1 mm.
Posterior root arteries	16	½ mm.
Posterior root veins	20 to 25	1½ to 2 mm.

Vessels of the Cord Substance.—The cord is suppplied by (1) central arteries which are branches of the anterior median, and by (2) peripheral arteries which come from the plexuses on the pia mater. These two systems have been called also the centrifugal and centripetal respectively. They are not absolutely independent, but are in a good measure so. The central arteries nourish chiefly the gray matter, the peripheral arteries the white. Both systems are made up of "end arteries," *i.e.*, they do not anastomose with each other. Neither the central nor the peripheral arteries are distributed in accordance with anatomical relations or physiological functions. Each cell group, for example, has a vascular supply from several sources.

The *central arteries* are given off from the branches of the anterior median at the bottom of the median fissure and number about two hundred, each spinal segment having six or seven. The accompanying central veins are small and their total capacity is less than that of the arteries, so that the central arterial pressure must be high, on account of the poor venous outlet (Kadyi). Some of the blood escapes by the peripheral veins.

The *peripheral* arteries pass into the spinal cord for the most part along the various connective-tissue septa. There they branch and supply chiefly the white matter. They supply the apex and some of the deeper substance of the posterior horns and Clark's columns. The arteries of the posterior septum are the largest and most numerous, often reaching to the gray commissure. The peripheral arteries are smaller than the corresponding veins (0.04 to 0.2 mm.). The relation is just the reverse, therefore, of that of the central arteries and veins. The peripheral arteries are small, and after passing into the cord branch into minute vessels which pass up and down and soon become capillaries. The central arteries, on the other hand, continue large, and run up and down some distance before they are subdivided into capillaries.

To sum up: The arteries predominate in total capacity in the anterior plexus and central arteries; the veins in the posterior plexuses and peripheral vessels. The central arteries are larger and longer than the peripheral. Hence the blood circulates more quickly and under greater pressure in the central gray of the cord. Conditions of enfeebled circulation would affect the posterior columns and roots more than the anterior and central parts of the cord.

CHAPTER XII.

THE DISEASES OF THE SPINAL CORD.

There are about thirty diseases which may be classified as belonging to the spinal cord. Most of these are organic in character and come under the head of inflammatory and degenerative or system diseases. Functional disorders referable to the cord alone are rare; while of organic diseases, those that result from injury and inflammation are the most common.

Etiology.—The causes of spinal-cord diseases can nearly all be formulated under the heads of injury, exposure, poisons, autotoxæmias, infections, and excessive functioning. Persons of middle life are the most predisposed, while heredity does not play an important part.

Symptoms.—The symptoms of all disorders of the nervous centres can be included under the heads of those of irritation, depression, and perversion. The principal irritative symptoms in spinal-cord disease are pains and paræsthesias of the back and limbs, hyperæsthesia and feelings of constriction around the waist, rigidity, spasms, exaggerated reflexes, and irritability of the visceral and vascular functions. The principal symptoms of depression and destruction are anæsthesia, ataxia, paralysis, wasting, and loss of power over visceral centres. The common form of paralysis in spinal-cord disease is paraplegia, in brain disease hemiplegia, in multiple neuritis quadruplegia. Symptoms of irritation and depression often accompany each other. The more superficial and meningeal the disease, the more are the symptoms irritative; the more central and myelonic the trouble, the less the irritation and the more the paralysis and visceral disturbance. Thus meningitis, meningeal tumors, and hemorrhages are extremely painful; while central myelitis is almost painless.

Pathology.—Inflammations of the meninges of the cord are not rare; the opposite is true of primary inflammations of the cord itself. As will be shown later, most of the diseases that used to be called chronic myelitis are secondary to injuries and softenings. Degenerative diseases of the cord, which include such affections as locomotor ataxia and progressive muscular atrophy, used to be called

"system diseases," because they affected certain long-fibre tracts or systems of cell groups. The name implies restrictions which are not justified in fact, and it can be retained only as a matter of convenience. Secondary degenerations alone are always systemic. The cord is relatively free from abscesses, hemorrhages, and tumors.

Diagnosis.—In making a diagnosis of spinal-cord diseases, one is most helped by a thorough knowledge of the cord functions. In no part of the economy do physiology and anatomy point out more clearly the path to the clinician.

Prognosis.—The spinal-cord tissue once destroyed can never be renewed, or only to a limited extent, and that as regards the nerve fibres, not the cells. It has considerable power of adjusting itself to damage; but, on the other hand, serious injury is likely to extend by the process of secondary degeneration. Functional diseases, vascular and nutritive disturbances, of the cord can never be so severe or chronic as to exclude the possibility of recovery.

The special diseases of the spinal cord are the following:

1. *Malformations:* Myelocele, meningo-myelocele (spina bifida), meningocele, heteropia, amyelia, micromyelia, macromyelia, double cord.

2. *Vascular Disorders:* Anæmia, hyperæmia, hemorrhage, endarteritis with aneurism, embolism or thrombosis, œdema. *Secondary* to these conditions are *softenings* and *sclerosis.*

3. *Inflammations:* Meningitis, myelitis, abscess. Secondarily, softenings, sclerosis.

4. *Degenerations:* Primary: locomotor ataxia, combined scleroses, hereditary scleroses, progressive muscular atrophy, and allied types.

5. *Tuberculosis:* Miliary and solitary.

6. *Syphilis:* Gumma, meningo-myelitis, vascular disease.

7. *Tumors.*

8. *Functional and toxic disorders.*

MALFORMATIONS.

Spina Bifida (Rhachischisis Posterior).

Spina bifida is a congenital hernia of the spinal membranes, and sometimes of the cord, through a cleft in the vertebra caused by absence of the vertebral arches. It is really a malformation of the vertebral canal rather than of the cord.

Etiology.—The condition is not very rare, about 1 child in 1,200 (French statistics) being affected. It is often associated with hy-

drocephalus or with some other defect in development, such as ventral hernia, imperforate anus or pharynx. Hereditary influence is sometimes a factor. It is a true developmental defect, and is not due to a primary dropsy of the cord, as was once taught. It occurs rather oftener in females.

Forms.— There are three varieties described:

1. Spinal meningocele is a condition in which the spinal membranes alone protrude into the sac.

2. Spinal meningo-myelocele is a form in which the membranes and cord both protrude.

3. Syringo-myelocele (hydrorrhachis interna) is a form in which the fluid is in the central spinal canal, and the inner lining of the sac is formed by the meninges and thinned-out spinal cord.

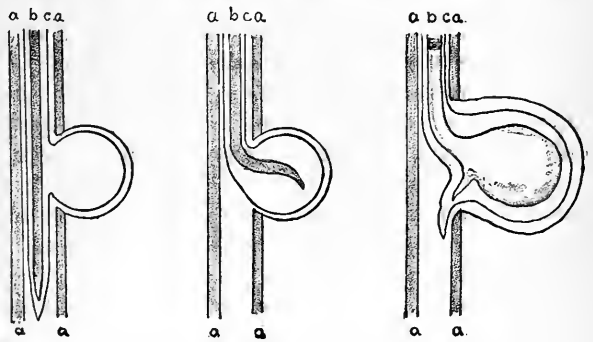


FIG. 114.—MENINGOCELE. MENINGO-MYELOCELE. SYRINGO-MYELOCELE. *a*, Vertebral walls; *b*, cord; *c*, membranes.

Anatomy.—The first two forms are the most common and are called hydrorrhachis externa. The fluid here lies in the subarachnoid sac, and hence the wall of the protruding cyst is lined with the dura and arachnoid. The nerves and cord protrude into the sac in two-thirds of the cases (forming a meningo-myelocele), but in some of these only a few nerves are found. These structures, when present in the sac, as in meningo-myelocele, lie on its *posterior and median surface.* They are attached to and form part of the wall. The spinal nerves therefore start from the wall of the sac and go back into the vertebral canal. The tumor contains cerebro-spinal fluid, and occasionally connective tissue and fat (Fig. 114). The external surface is often red and smooth, and there is sometimes a depression on its median surface where the cord is attached.

Symptoms.—Spinal bifida occurs almost always in the lumbar and sacral region, the reason being that the laminæ here are the last to solidify. Usually but two or three vertebræ are involved.

The tumor varies in size from 3 cm. (one inch) to 15 cm. (six inches) in diameter, and may have a broad base or be pedunculated. The outer skin is often glossy, or tough, thickened, or ulcerated (Fig. 115).

Children with spina bifida are usually feeble, badly nourished, and poorly developed mentally. Paraplegia occurs in half the cases, sometimes with anæsthesia and involvement of the sphincters. Talipes occurs quite often.

The *prognosis* is grave. Most subjects die unless treatment is applied, and even then the prospect is not very good. The prognosis is best for meningocele.

The *diagnosis* is easy. It is generally only necessary to exclude congenital tumors which happen to be located in the lumbo-sacral region. The most important question to decide is whether the cord and nerves are present in the sac. This may be assumed as probable if there is much paraplegia, anæsthesia, and sphincter trouble, and if there is a depression on the median external surface. The introduction of an insulated needle connected with an electric battery may be tried.

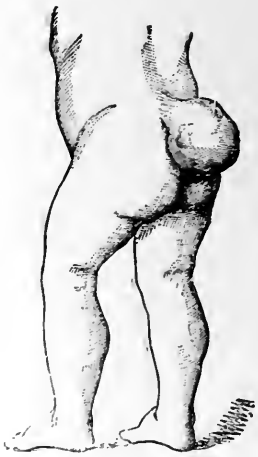


FIG. 115.—SPINA BIFIDA.

The *treatment* is strictly surgical, and then is of avail only in meningocele. At present, injections of Morton's fluid (iodine, gr. x.; potas. iodid., gr. xxx.; glycerin, ʒi. Dose, ʒi.) seem to be most successful. These injections should be made in the lateral portion of the sac, and the child should be kept on the back. Puncture and withdrawal of fluid with compression is not a justifiable operation. Ligaturing or opening and excising of the sac are dangerous, especially if, as is often the case, part of the cord and nerves lie in the sac. In recent years, surgical results have been more favorable and warrant serious consideration. No surgical treatment should be attempted, however, until two or three months after birth.

HETEROPIA is a rare malformation in which masses of gray matter are found in abnormal situations. A false heteropia may be caused (Van Gieson) by manipulation of the cord in its removal after death. The displaced masses consist of nerve cells or neuroglia.

AMYELIA or absence of the spinal cord can exist only when the brain is absent; but absence of the brain may occur without absence

of the cord. In amyelia the spinal nerves are usually present. Amyelic monsters cannot live.

Double Cord is a very rare defect and involves only part of the cord except in cases in which there is a double vertebral canal.

Double central canal is not rare. It usually involves only a part of the cord. The two canals are side by side.

Asymmetry of the cord, usually due to abnormality in the course of the pyramidal tracts, is not extremely rare.

Splitting of the cord and defects in development at special levels are occasionally observed.

Micromyelia is a condition in which the spinal cord is abnormally short or small in size, and is not a very rare anomaly. The normal adult cord has a diameter in its various parts of 6 to 9 mm. (dorsal), 8 to 11 mm. (upper cervical), 15 mm. (cervical swelling), and 12 mm. (lumbar).

SPINAL HEMORRHAGE (SPINAL APOPLEXY).

This general name may be given to (1) spinal meningeal hemorrhage or hæmatorrhachis, and (2) hemorrhage into the cord substance, or hæmatomyelia.

1. Spinal meningeal hemorrhage is far the most common form. It may be outside or inside of the dura, the former being rather oftener seen.

Etiology.—It occurs in newly born children and in adults, and is more common in men than in women. Injuries, falls, fractures of the spine are the most frequent exciting causes. Severe convulsions from epilepsy, eclampsia, tetanus, chorea, or strychnine may be a cause, also severe muscular exertion. Purpura and the blood states following malignant infectious fevers, bursting of an aortic or vertebral aneurism, and cerebro-spinal meningitis are rare causes.

Symptoms.—In small hemorrhages there may be no symptoms. In large effusions there are sudden, very severe pains in the back, extending into the limbs, numbness, tingling, hyperæsthesia, and muscular spasm, especially of the back muscles. Later there may be weakness or paralysis and anæsthesia, with disorder of the visceral centres. The symptoms reach their height usually in a few hours. Then amelioration may occur, followed by slow recovery or with symptoms of chronic meningitis. Rarely death occurs early from exhaustion.

Diagnosis.—A history of injury or childbirth, sudden onset of attack, with symptoms of pain and irritation which rather rapidly subside, point to extradural hemorrhage. In hæmatomyelia there are less pain and irritation, but more profound paralysis and anæsthesia. The same is true of crush of the cord from fracture or dis-

location. In tetanus there is a slower development of the symptoms, and trismus is present.

The *prognosis* is grave in severe cases, but if the patient survives three or four days the prospect of partial or nearly complete recovery is good.

The *treatment* is perfect rest in bed and the administration of remedies to move the bowels and relieve pain; leeches and other local applications are of doubtful value. It is of no use to give styptics except in purpura, when mineral acids or suprarenal extract may be tried. Later, one may give iodide and mercury and use blisters to the back.

2. Hemorrhage into the Substance of the Cord (Hæmatomyelia)—*Etiology.*—The condition is not very rare. It may be primary from disease of the blood-vessels or purpura hæmorrhagica; or it may be secondary to myelitis and tumors. Primary hemorrhage occurs sometimes in infancy, but usually in males between the twentieth and fortieth year. Injuries, overexertion, exposure, excessive coitus (Gowers), syphilitic disease of the blood-vessels, and convulsions are causes. The disease sometimes occurs in old people with degenerated arteries, which break and lead to a spinal apoplexy, just as occurs in the brain.

The *symptoms* develop rapidly, with at first feelings of numbness or weakness for one or two hours or longer. Then there is a sudden paraplegia, with anæsthesia or ataxia or both. The anæsthesia is often dissociated, there being loss of pain and thermic sense with retention of considerable tactile sense. The sphincters may be paralyzed; the urine has to be drawn. The reflexes may be abolished at first, but soon return and become exaggerated. There is considerable pain in the back. If the lesion is high up, the arms and thorax are involved. The acute symptoms begin usually to subside at the end of seven to ten days and the disease takes the character of a chronic myelitis. If improvement does not occur, evidences of acute myelitis appear and the patient dies.

Pathology.—The vessels involved are the central arteries, which supply the gray matter and are under relatively high pressure. The rupture of the vessel, when due to disease, is caused by a fatty degeneration of the coats or syphilitic endarteritis; miliary aneurisms, such as are found in the brain, rarely develop in the cord. Hemorrhage often precedes or begins a myelitis, of which it may be the cause or the result. The clot may be absorbed, leaving a cavity as in the brain; or the broken-down tissue may become the centre of a myelitic focus. The hemorrhage is usually single, but there may be several. Multiple capillary hemorrhages occur, but

usually only from asphyxia and convulsions. It is possible that some of the cases of disseminated myelitis occurring after infectious fevers start from small hemorrhages. Hemorrhage sometimes results from the invasion of the cord by a new growth, as in syringomyelia.

Diagnosis.—The points to be noted are the sudden onset without long premonitory symptoms, and the absence of fever followed later by gradual improvement. There is much less pain than in meningeal hemorrhage, and the dissociation of cutaneous sensations is very characteristic. In acute softening there is less of the dissociation of sensations and usually a more extensive paralysis. The disease is often mistaken for acute primary myelitis, which does in fact often follow it. Meningeal hemorrhage is more painful, and there is less paralysis, more spasm, and a more complete recovery later.

Prognosis.—This is often serious as regards life, and always serious as regards health. It depends on the extent and seat of the hemorrhages. Dorsal hemorrhages are more favorable, cervical the least.

Treatment.—Absolute rest, ice bags to the spine, and small doses of aconite given early are all that can be tried, except the use of symptomatic remedies. Treatment must be applied at once. The late treatment is the same as that for myelitis.

THE CAISSON DISEASE (DIVER'S PARALYSIS).

The caisson disease is the name given to a more or less complete paraplegia which occurs in persons who work in caissons or diving-bells, and which is brought about by the sudden return from a condensed air to the normal atmosphere.

Etiology.—Persons employed in caissons or bells work usually under a pressure of from one to four atmospheres, which means a pressure of from fifteen to fifty pounds to the square inch. Accidents rarely if ever occur when the pressure is not over one atmosphere, and they are also rare if the person has not been subjected to the pressure for at least an hour. Different persons vary in susceptibility to the effects of this change in the atmospheric pressure, and those unused to the work are more liable to be attacked. Naturally the disease is seen only in men, and during the working period of life.

The symptoms set in usually very soon after the patient has come out from the caisson, but they may be delayed for half an hour or more. They consist of intense neuralgic pains in the lower

extremities, often affecting especially the joints. There is at the same time epigastric pain. Nausea and vomiting and weakness in the lower limbs, amounting in some cases to absolute paralysis, very soon appear. There may be headache, dizziness, and sometimes even coma. If the paralysis is considerable, it is usually accompanied by anæsthesia. Disturbances in the sphincters, with retention of urine and constipation, may also be present. The symptoms vary very much in severity, from pain, weakness in the legs, and nausea, up to frightful neuralgic attacks and complete paralysis, motor and sensory. The upper limbs are rarely affected. In a few instances hemiplegia, however, has been observed. The disease lasts from a few hours up to several weeks. Death occurs in some of the very severe cases. The symptoms having reached their climax gradually ameliorate, and a complete cure is not infrequent. In some instances, however, the patient is left with a permanent paraplegia and the ordinary symptoms of a transverse myelitis.

Pathology.—When the patient is under atmospheric pressure in the caisson, the blood is driven from the surface of the body, and the internal viscera, including the brain and cord, are congested. The sudden change from the abnormal to normal pressure produces a rapid flow of blood from the internal organs to the periphery. The viscera not inclosed in bony cavities are enabled to relieve themselves of this congestion without much harm, but the circulation in the brain and spinal cord is less elastic; that in the spinal cord being less even than that in the brain. The result is that congestions and small hemorrhages ensue, producing a destruction of the nerve tissue. In other cases there seems to be a blocking up of some of the small vessels, with consequent softening of different portions of the cord and to a less extent of the brain. It is supposed that one element in producing the morbid phenomena is the escape of oxygen and carbonic-acid gas from the blood into the tissues or into the blood-vessels. This mechanism, however, has not been proven. It will be seen, however, that on the whole the sudden change in atmospheric pressure leads to vascular disturbances with rupture or obliteration of blood-vessels, with consequent destruction and necrosis of tissue. Following this is a reactive inflammation producing the phenomena of an ordinary acute myelitis.

The treatment is largely prophylactic. The workmen engaged in the occupation should be carefully selected and should accustom themselves to their work. They should spend a longer time in coming out of the caisson. If symptoms supervene, it is recommended that they be put back under a slight atmospheric pressure at once until these symptoms disappear. When the disease has developed,

it can be treated only by symptomatic remedies. The patient should be kept quiet, and given, if necessary, hypodermics of morphine. Dr. A. H. Smith recommends the use of ergot. Later on, the various neuralgic and paralytic symptoms may be treated on the same principles as those employed in myelitis.

SPINAL HYPERÆMIA, ACUTE AND CHRONIC.

Etiology.—Acute spinal hyperæmia is produced by sexual excesses, violent physical exertion, suppression of menstrual discharges, and certain poisons like strychnine. It occurs also in the first stage of acute inflammatory diseases.

Chronic spinal hyperæmia is, so far as is absolutely known, a very rare condition. Chronic hyperæmia of the membranes may be the residuum of a meningitis or of injury, and these are probably the most common causes. As to the causes of the chronic hyperæmia of the cord substance itself independent of other diseases, we can say nothing definitely.

The symptoms of acute spinal hyperæmia are feelings of heaviness and weight in the limbs and around the loins, numbness, creeping sensations and actual neuralgic pains, weakness of the lower limbs, with twitchings of the muscles. There may be also some disturbances in the sphincters, though of this one can speak less certainly. The symptoms are nearly always confined chiefly to the lower limbs. The statement that they are increased by lying on the back and ameliorated by lying on the face is not always true, since posture, unless greatly prolonged, influences but very slightly, if at all, the circulation in the spinal cord.

The symptoms of chronic spinal hyperæmia, when this hyperæmia involves the meninges chiefly, are probably somewhat identical with those of spinal irritation. They will be described under that head.

Pathology.—The circulation of the blood in the spinal cord, as has been shown in the article on anatomy, is one which it is difficult to disturb, but which, once disturbed, is rather slow in being brought back to its normal condition. Thus violent activity of the heart and great increase in the arterial pressure, and the opposite conditions of weakened heart and lowered arterial tension, appear but little to modify the spinal functions. Hence it is unlikely that the large number of clinical symptoms that have at times been attributed to hyperæmia of the spinal cord, or rather to disturbances in the circulation of the spinal cord, have really been due to that cause.

Treatment.—The treatment of spinal hyperæmia consists in the application of cups to the back, quiet in the horizontal position,

preferably upon the side or face, ice and counter-irritants to the spine, morphine and bromides internally. In the more chronic cases muriate of ammonium, iodide of potassium, and the galvanic current may be used. The use of ergot, which has been recommended, in my experience has been found of little or no value.

SPINAL ANÆMIA.

Even less is known in regard to the etiology and symptomatology of spinal anæmia than of spinal hyperæmia. Undoubtedly severe hemorrhages or diarrhœal discharges, and an aortic obstruction which cuts off the circulation of the blood from the spinal cord, will produce a spinal anæmia, and when this is severe the functions of the cord are nearly abolished. But practically we do not know of any causes which produce an acute or chronic anæmia leading to serious and prolonged disturbances in the spinal functions, aside from diseases of the arteries of the spinal cord themselves, such as occur in advanced life. In the most profound anæmias, which must affect equally the spinal cord with other organs, very little special disturbance of this organ can be discovered. Here, too, the supposed test of improvement on lying on the back is, in the writer's opinion, a fallacious one. It has been customary to associate with spinal anæmia a class of symptoms characterized by pains in the back of the nature of spinal irritation, weakness of the legs amounting to paraplegia—a type of symptoms that has been called spinal concussion, but it is impossible in the present stage of science to say that a spinal anæmia actually underlies and causes this condition.

INFLAMMATION OF THE SPINAL MEMBRANES (SPINAL MENINGITIS).

The meningeal inflammations are:

External meningitis. Internal meningitis.	Affecting the dura mater.
Leptomeningitis.	Affecting the pia mater.
Hypertrophic pachymeningitis.	Affecting both membranes.

External Meningitis, Pachymeningitis Externa ("Compression Myelitis").

Etiology.—The disease is rare, and always occurs secondarily to some other morbid process. This process is in most cases tuberculosis and causing caries of the vertebræ. Other causes are suppurative inflammation in the neighborhood of the vertebræ, psoas abscess, purulent pleurisy, peritonitis, and puerperal pyæmia. When the disease arises from inflammations in the visceral cavities, it is thought to be caused by an ascending neuritis.

Symptoms.—The symptoms are those of irritation of the motor and sensory roots; later, compression of them and of the spinal cord, local pain in the back, radiating pains, tenderness, hyperæsthesia, twitching, paresis, paraplegia, exaggeration of reflexes, and involvement of the sphincters. Anæsthesia occurs in severe forms.

The disease, when chronic, may extend to the other membranes and cord, causing what is termed "compression myelitis."

Pathological Anatomy.—The inflammation, if acute, is generally a fibro-purulent one, this being the form usually caused by vertebral caries. The dura mater is covered by a layer of caseous, semi-solid matter, often very thick and most extensive posteriorly. It involves the dura vertically for several inches. In chronic forms the deposit is made up of connective tissue and the cord is compressed. In purely suppurative forms the cellular tissue outside the dura is infiltrated with pus throughout a great part of the canal.

The *diagnosis* is based on the presence of the primary local disease, the kyphosis, the radiating pains, and tenderness, and by the combination of motor and sensory irritation and paralysis.

The *prognosis* is generally bad, because the original disease is a serious one. Still, surprisingly good results are often obtained when the disease is taken early.

The *treatment* consists in attention to the local caries or inflammatory focus. It is therefore purely surgical. Some kind of jacket is almost always indicated.

Internal Meningitis, Pachymeningitis Interna, Hemorrhagic and Hypertrophic.

Inflammation of the inner surface of the dura mater occurs in two forms—the hemorrhagic and the hypertrophic. As the latter is generally but a sequel of the former, I shall describe the two together under the head of *hypertrophic pachymeningitis.*

Etiology.—The disease occurs almost always in adult life. A few cases have been reported in children (Gibney). It usually affects males. Syphilis, alcoholism, exposure, and trauma are exciting causes, the first and last being by far the most important factors in the disease. My own experience is that the disease is always a syphilitic process.

Symptoms.—The disease begins gradually with symptoms of irritation (irritative stage). The patient suffers from pain and stiffness in the neck. The pains radiate up to the occiput and down the back; numbness, prickling, and pain are felt in the arms, more in one than the other. The pains exacerbate and are worse at night. Stiffness and cramps may affect the arms. Nausea and vomiting sometimes occur.

After five or six months symptoms of paralysis appear (paralytic stage). The arms are affected. They become weak, atrophy occurs,

associated with contractures and rigidity. There is still pain, and in addition anæsthesia, hyperæsthesia, and trophic changes occur. Later, paraplegia, with rigidity, exaggerated reflexes, and spinal trepidation develop. The patient becomes weaker, and finally dies of exhaustion or from some intercurrent trouble. The disease sometimes takes what is called the *peripheral type*. Then the symptoms are more localized in the extremities. Usually it is of the *cervical type* and presents symptoms as described above. In either form the disease is a chronic and painful one.

Pathology.—The disease starts as a hemorrhage upon the surface of the dura. This leads to a chronic inflammatory process, new hemorrhages occur, and a fresh inflammatory deposit is made until the cord is finally encircled and compressed by a dense connective-tissue mass, which involves the pia and to some extent the cord substance. The process is analogous to that of cerebral pachymeningitis hæmorrhagica. The cervical region is usually attacked. In other cases the lesion is a syphilitic gummatous process.

Prognosis.—A few cases have been reported practically cured. More cases terminate in death. Sometimes, however, the disease comes to a standstill for a long time.

Diagnosis.—This must be made from tumor, myelitis, Pott's disease, wryneck, and progressive muscular atrophy. The history of injury, the slow progressive course, and the localization of the symptoms, their bilateral character and the pain, give the most help. It is not always possible to exclude a tumor. In spinal tumor the symptoms at the beginning are more sharply localized. They develop more rapidly and the course of the disease is shorter than in meningitis. Tumor though rare is really much more common than pachymeningitis. It is probable that some cases described as this were really forms of syphilis of the cord.

Treatment.—The not rare syphilitic origin of the disease must be borne in mind. Counter-irritants, electricity, hydrotherapy, and symptomatic remedies for the pain and spasms are indicated. It is possible that surgery may help these cases.

Acute Spinal Leptomeningitis (Inflammation of the Pia Mater of the Spinal Cord).

Etiology.—This is a rare disease, occurring alone, but is common in conection with disease of the cerebral pia mater.

Children are oftenest affected; and among adults, males. Alcoholism predisposes to it. The disease is always secondary to an infection with or without a traumatism. The infections are tubercle, syphilis, typhoid fever, and various pyogenic microbes. Extension of inflammation from neighboring parts and surgical operations are occasional causes. The cases attributed to rheumatism, exposure, insolation, are in reality due to some infection, and the virus of cerebro-spinal fever sometimes attacks the cord alone.

Symptoms.—The disease begins with pain in the back, radiating along the nerves. There are usually a chill and some fever. The pulse may be fast or slow. The pain increases, and is accompanied by dorsal tenderness and rigidity of the muscles of the back, amounting sometimes to opisthotonos. The skin is very hyperæsthetic and the reflexes are at first increased. There is constipation, and sometimes retention of urine. After a time symptoms of paralysis come on, with anæsthesia and retention of urine. The patient becomes weaker, bedsores may form, and death from exhaustion follow. The disease lasts from a few days to several weeks.

The dominant symptoms in the first stage are those of irritation, viz., pain in the back and along the nerves, hyperæsthesia, and muscular spasm. In the second stage, paralysis, atrophy, and anæsthesia.

In the *tuberculous* form of meningitis the symptoms come on more slowly. In *septic* meningitis the symptoms are of the severe and typical kind described. In meningitis from other infections the symptoms are not so severe, as a rule.

Pathological Anatomy.—Acute leptomeningitis shows a somewhat different exudate according to the nature of the infecting microorganisms. The common form is the suppurative exudate, which may be due to the streptococcus pyogenes and other pyogenic microorganisms, and to the pneumococcus. The purulent matter is usually distributed along the whole length of the cord, but more posteriorly and often more in its lower portion. It may also be localized chiefly at certain levels. The spinal fluid is increased in amount. The arachnoid, the inner surface of the dura, and the tissue of the cord itself are usually involved. If the disease lasts several weeks, the purulent matter is absorbed in part and an increase in connective tissue takes place, binding the dura, arachnoid, and pia to the cord. The nerve roots are surrounded and compressed by the inflammatory product.

In tuberculous meningitis there is less exudate. It is more of a fibrinous character, and grayish in appearance. Tubercle granulations are seen distributed over the pia, arachnoid, and inner surface of the dura. Simple exudative meningitis rarely occurs. The inflammatory process often ends in a production of new connective tissue and sometimes the establishment of a chronic leptomeningitis.

The *diagnosis* must be made from myelitis, tetanus, rabies, rheumatism of the dorsal muscles, gonorrhœal rheumatism, and strychnine poisoning. In myelitis there is relatively little pain and much paralysis; in tetanus there is trismus, fever is absent, and there is a history of trauma.

Tuberculous meningitis comes on more slowly, is rarely spinal alone, and there may be evidence of local tuberculosis elsewhere.

The *prognosis* is not good, but is especially bad in tuberculous meningitis and in cases with high fever, severe pains, and early paralysis. Chronic meningitis sometimes remains after the acute symptoms subside.

Treatment.—This consists first in perfect rest and quiet; leeches should be applied along the spine, then hot poultices or ice bags; opium is to be given for relief of pain; mercurial purges and small doses of iodide of potassium or sulphate of magnesium may be given at short intervals. Later, blisters and counter-irritation and lukewarm baths are indicated.

Chronic Leptomeningitis and Meningo-myelitis.

Etiology.—This disease, which used to be often diagnosticated, is now believed to be rare, and always secondary to an acute process, such as a cerebro-spinal meningitis, or to syphilis and perhaps chronic alcoholism. It occurs oftenest in adults and in males. Trauma, and especially concussion of the spine, used to be thought a frequent cause, but in most of such cases the trouble is simply a hyperæmia or else neuralgic and functional.

The *symptoms* gradually develop after an acute meningitis or an injury, and they are the same in character as those of the acute process. There are pain in the back, increased on movement and radiating about the trunk and down the limbs; tenderness along the spine, stiffness of the back, twitching and spasms in the limbs with some weakness, and later some paralysis, wasting, and anæsthesia, with weakness of the bladder. These paralytic symptoms, if severe, however, mark an invasion of the spinal cord. Cutaneous eruptions, such as herpes, may appear. The symptoms run an irregular course, with periods of improvement. They always become less when the patient rests.

Pathological Anatomy.—The inflammation consists of a proliferation of connective tissue (productive inflammation of Delafield). The result is a thickening and opacity of the pia mater and arachnoid. The dura mater may be involved, but only in severe cases. The process may invade the spinal cord, causing an "annular" or ringlike sclerosis, from which the disease extends into the cord in wedge-shaped masses at various parts, causing eventually an involvement of fibre systems and secondary degenerations up and down. This somewhat rare terminal condition is called meningo-myelitis. In syphilitic meningitis the lesion usually involves only part of the spinal cord, usually some of the dorsal segments. Here there is

the characteristic exudate of syphilis, not of a simple proliferative inflammation.

The *diagnosis* must be made from spinal irritation, locomotor ataxia, myelitis, vertebral caries, and tetanus. In spinal irritation there are not the rigidity, severe radiating pains, twitchings, atrophy, or paralysis; and neurasthenia or hysteria exists. In locomotor ataxia the knee jerk is lost, there is ataxia, and there is little paralysis, nor is there local tenderness over the spine.

In vertebral caries the pain and tenderness are much more localized, and there is spasmodic fixation of the trunk. The pain is more continuous and dull, and is increased by lateral pressure and lessened by extension. There is usually also some deformity. If compression occurs there is exaggeration of the reflexes and paraplegia, without much anæsthesia. After all, however, with Pott's disease there may be a local meningitis.

Treatment.—As chronic meningitis is usually the product of syphilis, or the relic of traumatism or of an acute process, the indications for treatment are simple. Rest is the essential thing. With this can be combined the systematic and persistent use of counter-irritants. The hot iron is usually best, because its wounds heal so quickly. Cupping is also useful if done vigorously and often. Internally, iodide of potassium, small doses of bichloride of mercury, nitroglycerin, and digitalis may be given; also the salicylates and ergot. Both the latter drugs should be employed in large doses, if at all. Electricity in the form of galvanism, and cocaine by local injection or cataphoresis, may be tried. Splints and plaster jackets are of much service in some cases.

CHAPTER XIII.

MYELITIS—INFLAMMATION OF THE SPINAL CORD.

Myelitis is an inflammation of the spinal cord. It may be *acute*, *subacute*, or *chronic*.

It may affect the anterior horns chiefly, when it is called *anterior poliomyelitis;* or both the gray and white matter, when it is called *diffuse myelitis* and *transverse myelitis*. The forms are still further divided, in accordance with their location, into cervical, dorsal, lumbar, and disseminated myelitis. Myelitis is given different names also in accordance with its cause. Thus we have *hemorrhagic myelitis*, a form in which the initial process is due to or associated with a hemorrhage; *compression myelitis*, due to vertebral caries; *septic* or *purulent* myelitis or abscess of the cord; and *tuberculous* and *syphilitic* myelitis.

For practical purposes the following classification is sufficient:

Acute myelitis:	
With exudation and necrosis.	1. Anterior poliomyelitis.
	2. Transverse, or diffuse, or disseminated myelitis.
With suppuration.	3. Abscess of cord.
Chronic myelitis:	
With necrosis and proliferation.	4. Chronic anterior poliomyelitis.
	5. Transverse myelitis.
	6. Compression myelitis.

Acute Transverse Myelitis (Acute Softening of the Spinal Cord.)

Acute, diffuse, or transverse myelitis represents several different pathological processes. It may be a local infection causing exudative inflammation, with more or less necrosis resembling acute anterior poliomyelitis. The difference in the symptoms here depend only on the severity of the infection, its rapidity of course and extent. It may be initiated or accompanied by a hemorrhage or softening from thrombosis of arteries, and the latter is often the case. Nor can we clinically distinguish between the cases due to a pri-

mary infection and those due to a hemorrhage and softening. Various pathogenic microbes have been found, but they are not always present even in real infections; because the microbe after starting its work is absorbed and disappears.

Hence acute transverse myelitis may mean either an acute inflammatory process or an acute softening.

Etiology.—Predisposing causes are a neuropathic constitution, the male sex, early adult life, occupations calling for exposure, and muscular strain. The exciting causes are exposure to cold, blows, falls, fractures, strains, extension from neighboring organs, syphilis, infective fevers, and septic infection. *Injury* is frequently an apparent cause. But most of these causes produce primarily mechanical destruction, hemorrhages, or thromboses, and the inflammation is secondary.

Symptoms.—*Prodromal* symptoms are rarely present, but there may be slight paræsthesias or pain in the back and limbs. Sometimes there is a chill, and in a few instances convulsions have been noticed.

The *initial* symptoms consist of feelings of numbness, usually in the feet and legs, which seem heavy and weak. Some pain may be felt about the back. The patient soon finds that he cannot walk easily, that he moves his legs with an effort and that they feel stiff. In a few hours or perhaps not till one or two days, a paraplegia with anæsthesia has developed, and if the lesion is in the cervical cord the arms are paralyzed also. All these symptoms may come on in the daytime or during sleep. Retention or incontinence of urine and constipation are early symptoms. There may be some fever.

Symptoms of the Attack.—If the patient is examined when the malady reaches its height, it will be found that he cannot walk or stand, but can move his legs a little. He complains of a sensation like a band around his waist or at the level of the spinal lesion (girdle symptom). His legs feel numb and heavy, but there is little pain and no tenderness. Anæsthesia to touch, pain, and temperature exists in various degrees on the limbs, as high up as the lesion. The anæsthesia, if not total, is greatest to touch, next to temperature, and least to pain. He has vesical anæsthesia; the urine is retained and he has to have it drawn. The bowels are constipated, but if enemata are given the fæces may pass away without his knowledge, owing to rectal anæsthesia. If the lesion is lumbar, there is abolition of the sexual power; but if dorsal or cervical, strong erections may occur without the patient's feeling them. When the lesion is above the lumbar cord, also, the bladder may automatically and forcibly contract and expel the urine. In time the bowels re-

gain some power. The paralysis in the limbs affects the flexors of the feet and legs more than the extensors. The patient can push down his limbs better than he can draw them up.

The temperature of the limbs for a few days is raised, but after this it falls a few degrees below normal. The skin becomes rough, cold, congested; and excessive perspiration may take place. The general bodily temperature is usually normal throughout the disease, but in some cases a fever develops of 102° to 104° and continues. The prognosis is then bad.

Bed-sores may develop early, within a few days or weeks. They appear oftenest upon the buttocks and heels, and are due to trophic disturbance, combined with pressure and pyogenic infection of the parts.

If the lesion is lumbar, the tendon and skin reflexes are much weakened and the paralysis is somewhat flaccid. The muscles also tend to waste and show degenerative reactions. If the lesion is dorsal, as is more often the case, the reflexes are present, and after a time become exaggerated; there is ankle clonus and a flexor response on irritating the soles of the feet; contractures and spasms develop; the legs become drawn up and deformities are produced. If the lesion is so complete as entirely to cut across the cord, there may still be some excessive muscular tension, but the reflexes will be abolished (Bastian). When the cervical region is attacked the arms are involved as well as the legs, and generally to a severer extent. There may now be also unequal dilatation of the pupils from involvement of the cilio-spinal centre; and optic neuritis from some cause has been known to occur. In extensive involvement of the upper part of the cord there may be paralysis of the intercostal muscles and disturbance of the heart's action.

The disease, having in a few days reached its height, usually remains stationary for a few weeks, and then, should the patient live, improvement slowly sets in. In some cases evidences of extension upward or downward occur (ascending or descending myelitis); the symptoms become more severe, and in a few weeks, or oftener months, death occurs.

As improvement begins, a return of sensation is first noticed (one to six months); this is followed by return of more or less motion (six to eighteen months). Spasms and contractures now develop, owing to a descending degeneration. A certain amount of ataxia from ascending degeneration, with a little anæsthesia of the skin, may remain, so that, if the patient has sufficient motor power to walk somewhat, he presents many features of "ataxic paraplegia" (see p. 305).

Some improvement may be expected for from one to two years. A few cases get almost entirely well. The majority become more less or paraplegic and bedridden, in which condition they are regarded as cases of chronic myelitis.

Pathological Anatomy.—The early changes found in acute myelitis are those of inflammation, hemorrhagic extravasation, and softening. Often it is impossible to say whether the primary process was inflammatory or due to a hemorrhage or softening.

Macroscopically, the cord at the affected part appears soft, swollen, and either red and hyperæmic or pale and anæmic. In rare cases no change is apparent to the naked eye. In later stages the parts are white or gray, shrunken, and hard. The cord may be reduced to a thin shred. The meninges about the affected parts are often thickened, inflamed, and adherent.

Microscopically, if the process is primarily inflammatory we find intense congestion, distended blood-vessels, emigrated white blood cells in great number, especially in the perivascular spaces, swollen axis cylinders and œdematous swelling of the myelin sheath, red blood cells, cells filled with fat granules known as compound granular corpuscles, or Gluge's corpuscles. These are leucocytes which have taken up fat granules. A peculiar form of cell, stellate in shape, known as spider cells or Deiter's cells, may be seen. They are proliferated neuroglia cells. The nerve cells lose their granular or reticular appearance, become homogeneous, swell up, fat granules appear in them, and a peculiar coagulation process attacks the cell and body. The cell processes retract, become thick, and finally drop off. Vacuoles are sometimes seen in the nerve cells; but these are very rare if the tissue is properly preserved. Besides the above evidences of vascular and connective-tissue activity and of cell destruction, one sees granular matter, broken-down nerve fibres, and other evidences of nerve disintegration. Pigment or extravasations of blood, and in later stages bodies resembling starch granules (corpora amylacea), may be seen. They are probably modifications of the myelin substance. If the lesion is primarily softening there may be much congestion, but the blood-vessels are less distended and fewer white cells are found in the perivascular spaces. The connective-tissue cells are less numerous. The nerve cells are swollen, glassy, and stain badly. There is a great deal of granular matter and nerve detritus. It is thought that in inflammatory swelling of the nerve cells they take up the carmine stain, but in degenerative changes they do not.*

* In the different stages of acute degeneration, the nerve cells, particularly those of the anterior horns, show various appearances. These have been

The connective-tissue changes are most noticeable in the white matter. The axis cylinder and myelin sheath are here often so disintegrated that in thin sections they drop out, leaving holes in the section and giving it a vacuolated appearance. The lesion, whether destructive or inflammatory, may extend up or down the cord, often in the course of certain tracts. Sometimes a destructive process extends a long way through the central part of the cord (perforating necrosis).

After three or four weeks, if the patient lives, the process of absorption and cicatrization begins, and secondary degenerations are seen. The granular and fatty matters and leucocytes disappear, the blood-vessels are thick-walled and distended but less numerous, connective tissue gradually takes the place of the destroyed nerve cells and fibres. The axis cylinder is the last to be destroyed and has the greatest power of recuperation. The nerve cell, if once destroyed, is never developed again. The cord at the point or points inflamed or softened becomes reduced to a mass of connective tissue containing, perhaps, a few nerve fibres and cells. In this stage the condition is one that is known as chronic myelitis. In some cases a cyst is formed in the affected region.

In fatal cases the inflammation and softening continue; fresh areas of cord are involved, much meninegal exudation takes place and finally death occurs. The process may in very rare cases be still more acute, suppuration and abscess occur, and here death also rapidly ensues.

The inflammatory and softening processes above referred to are described in accordance with their appearance as red softening, yellow softening, or white softening. A form of so-called inflammation known as inflammatory œdema is also described. It is an abortive inflammation, "a lymphatic congestion," analogous to vascular congestion.

The *diagnosis* of acute myelitis must be made from hemorrhage, acute embolic or thrombotic softening, acute ascending paralysis, multiple neuritis, meningitis, and meningeal hemorrhage, and hysterical, or functional paralysis. Spinal hemorrhage comes on suddenly and is usually not attended by fever. If meningeal, it is attended with pain. Acute softening cannot always be distinguished from acute myelitis, of which it is probably the starting-point in many

described by Friedmann as. 1, homogeneous swelling, hyaline swelling; 2, sclerosis; 3, simple atrophy; 4, fatty and molecular decomposition; 5, cloudy swelling. The degeneration begins in the cell body and then involves the processes and the nucleus; finally the whole cell is involved. Nerve cells do not often undergo the acute general death called coagulation necrosis (Friedmann), though the process of sclerosis, so called, resembles it somewhat.

cases. In softening there is *no leucocytosis*, the process is slower, there is less pain and the dissociation of cutaneous sensations is less marked. In acute ascending paralysis the disease is progressive, there are no involvement of sensation, no atrophy, and little change in the electric irritability. In multiple neuritis the onset is slower, there are more pain and local tenderness and sensory disturbance, and the bladder and rectum are rarely involved. In meningitis there are pain and tenderness in the back and limbs, rigidity, cramps, a little paralysis, and no bladder trouble. In hysterical paraplegia there are no marked atrophic changes, but little spasm or rigidity, no electrical changes, and the stigmata of hysteria may be found. The sensory disturbances are variable and somewhat characteristic (see Hysteria), and the knee-jerks are not greatly if at all exaggerated.

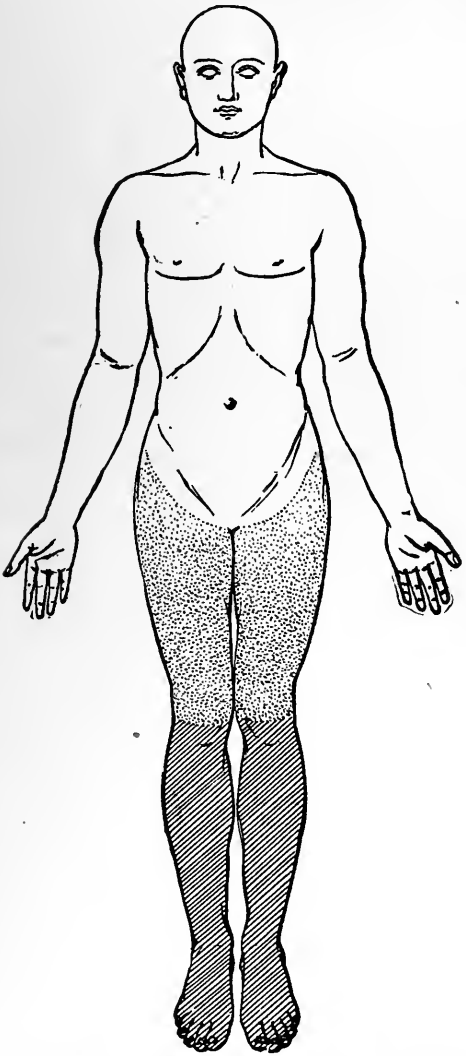


FIG. 116.—ACUTE TRANSVERSE MYELITIS OF LUMBAR CORD, showing distribution of anæsthesia. Area in lines=total anæsthesia and analgesia. Dotted area =analgesia only.

The diagnosis of the *location of the lesion* is made by studying the height of the anæsthesia, the skin reflexes (see p. 47), and the distribution and extent of the paralysis. There is often a differentiation of the anæsthesia as shown in Figs. 117 and 116. Lesions of the lumbar region involving the gray matter cause very complete paraplegia with sphincter troubles and degenerative electrical reactions. Lesions in the dorsal cord cause

a less complete paraplegia, but, owing to the secondary descending degenerations of the lateral column, rigidity, exaggeration of the deep reflexes, and contractures occur. Lesions of the cervical cord cause paralysis of the arms, with degenerative reactions in the muscles. The paraplegia is spastic and there is not much muscular wasting. If the lesion cuts entirely through the cord the limbs are paralyzed and may be somewhat rigid, but the deep reflexes are absent.

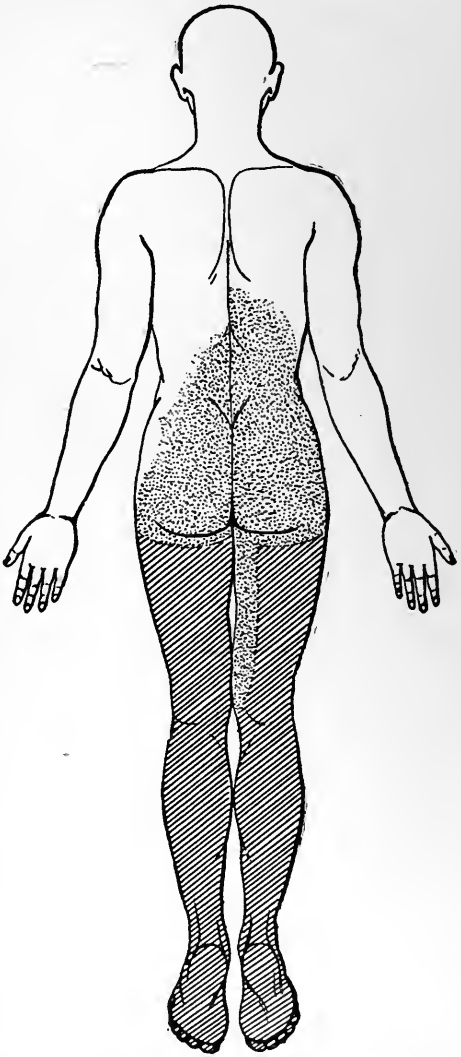


FIG. 117.—ACUTE TRANSVERSE MYELITIS OF LUMBAR CORD, showing distribution of anæsthesia. Area in lines=total anæsthesia and analgesia. Dotted area=analgesia only.

Prognosis.—The prognosis is worse the more complete and extensive the paralysis. It is worse in serious motor paralyses than when sensation is chiefly involved. It is best in dorsal myelitis and worst usually in cervical myelitis, other things being equal. Bedsores and slight fever are unfavorable signs; so also is severe involvement of the bladder and rectum. Recovery of sensation gives good hope of recovery of some motion. Total absence of recovery of sensation and motion after six months is very unfavorable. Improvement may be expected up to eighteen months after the onset, and in some cases even longer. In compression myelitis there is more chance of recovery than in the other forms. Syphilitic cases have a more favorable prognosis.

Treatment.—In the attack, the patient must be put to bed;

leeches or wet cups should be applied to the spine, diaphoresis should be promoted, small doses of aconite and nitroglycerin should be given, and a calomel purge administered. The bladder should be watched. After a week, moderate doses of iodide of potassium should be given. After about three weeks, if there is no fever, electricity may be applied carefully and strychnine administered in small doses. Bed-sores should be guarded against by the use of water beds or cushions, absorbent cotton, bathing the parts with alcohol and weak solutions of tannin. Infusion of buchu, boric acid, and tincture of hyoscyamus will often help the bladder disturbance. The frequency of syphilis as a cause of myelitis (nearly one-half the cases) should lead to the persistent use of mercurial inunctions and of iodide for a long time. After acute symptoms subside, tonics such as arsenic, iron, and strychnine in small doses may be given. Suspension may be tried carefully; mechanical appliances may be used to help the rigid limbs; lukewarm baths, douches, and massage are helpful to some extent.

Chronic Myelitis (including Transverse, Diffuse, Disseminated, and Compression Myelitis).

Chronic meylitis is the name given to a disease characterized by a chronic inflammation of the spinal cord and to the chronic reparative processes which follow acute inflammation, injury, and softening. Chronic myelitis is usually a mixture of inflammatory, reparative, and necrotic processes.

Forms.—Different names are given to chronic myelitis in accordance with the part of the cord affected. Usually the disease affects only certain levels, and then it is called *transverse myelitis.* More rarely it is *diffuse* or *disseminated*, *central* or *marginal.* When caused by pressure from vertebral disease, it is called *compression myelitis.*

Etiology.—The disease may be either *primary* or *secondary.* The *primary* form is somewhat rarer. It occurs chiefly in adults and in early and middle life, and much oftener in males. Exposure, shocks, infectious fevers, lead, and syphilis are the chief causes, but above all syphilis. Syphilis causes it by producing arterial disease and by setting up a specific inflammatory infiltration. *Secondary* chronic myelitis is a rather common form. It is really only the later stage of acute myelitis, softening, hemorrhage, or injury.

Meningitis may extend and cause a meningo-myelitis. A neuritis may possibly ascend and cause myelitis, but such cases, if they occur, are very rare. Compression myelitis is usually a slowly de-

structive, not an inflammatory process, and it begins as a meningitis of the dura mater.

Among 67 personal cases there were 61 men and 6 women. The causes that can be assigned were: Syphilis 23; injuries 12; exposure 3; acute infection 6, of which 3 followed grippe, 1 typhoid and 1 meningitis, 1 mumps. Two were due to caisson disease; 4 were of arterio-sclerotic origin occurring in the aged, and 6 were due to tuberculosis. A study of the age shows that in the decade between thirty-one and forty there were 23 cases; twenty-one and thirty, 14 cases; forty-one and fifty, 16 cases; fifty-one and sixty-four, 73 cases; eleven and twenty, 2; under ten, 2. Practically all the cases occur between the ages of twenty-one and fifty, and most of them between the ages of thirty-one and forty. Those cases occurring in the extreme of life are due to senile arterial changes or to tubercle or injury. There is not much difference in the age relations of the cases of syphilitic origin and those due to other causes. Between twenty-one and thirty, 5 out of 14 were of syphilitic origin. Between thirty-one and forty, 9 out of 23 were of specific origin, and syphilis may fairly be said to cause over one-half the cases of non-traumatic and non-tuberculous myelitis of adult life.

Symptoms.—When the disease begins primarily as a chronic affection the symptoms are as given below. And since nearly all the cases are of syphilitic origin, the description of primary chronic myelitis is practically that of syphilitic spinal paralysis.

The patient notices that his legs seem heavy and easily get tired; prickling and numb sensations are felt in the feet; occasionally a little pain develops in the back or there is a sense of constriction about the trunk. The legs are stiff, and tests often show that the reflexes are exaggerated, with ankle clonus and flexor response (sign of Babinski). There is but little wasting of them, however. The sexual power declines; the bladder gives some trouble, there being a tendency to retention; the bowels are constipated. After a few weeks or months there is a partial paraplegia, with rigidity of the limbs and exaggerated reflexes. Some anæsthesia exists, and occasional pain, which is not severe and is felt more in the back than the legs.

The muscles have now wasted somewhat, but show no decided changes to the electrical current. The bladder becomes more involved, the urine has to be drawn, it is often alkaline, and unless care is taken cystitis develops. The patient is still able to walk, but he does so with a stiff, shuffling gait which is characteristic (Fig. 118). The disease may show signs of slowly extending up and down, more often up. The arms become involved; weakness and stiffness, with some wasting, anæsthesia, and pain, develop, or the disease may cease its progress and the patient remain partly paralyzed for years. The general health during the course of the disease

deteriorates slowly; the patients often become anæmic and have an unhealthy pallor. Eventually the paraplegia becomes complete, the patient is bedridden, the legs are atrophied, contractured, and rigid, with more or less anæsthesia. Cystitis and nephritis develop, and the patient dies from these or other intercurrent diseases.

Chronic secondary myelitis, which is the form often seen, presents eventually much the same picture as that just described. In this type, however, the symptoms are worse at first, then improve or regress, then become stationary, and finally grow worse.

FIG. 118.—ATTITUDE IN CHRONIC MYELITIS.

Symptoms of the Different Forms.—The usual type of chronic myelitis is the transverse dorsal or dorso-lumbar, and this gives symptoms as above described. It is, as stated, usually a *syphilitic spinal disease.* If the lumbar region is affected there are more paraplegia, wasting, and involvement of organic centres. If the myelitis is cervical the arms are involved, there may be pupillary changes, and the respiratory muscles are partly paralyzed; the paraplegia is not so complete and the disturbances of sensation are likely to be more varied.

Compression myelitis, so called, is usually only a compression atrophy. It is due, as a rule, to vertebral caries, but its cause may be a spinal tumor, aneurism, and pachymeningitis. Compression myelitis is distinguished from other forms by its slow onset and the presence at first of irritative or "root" symptoms. The patient suffers from pain and tenderness localized at a certain point in the spine. The pain radiates about the trunk or down the limbs and is increased on movements. At about the same time some motor weakness develops, usually in the form of paraplegia. The muscles waste but slightly. The reflexes are exaggerated; twitchings, spasms, and contractures finally occur, and there is developed a spastic paraplegia or quadruplegia. With this there is usually some anæsthesia, though it is not complete. The disease is oftenest in the dorsal or lower cervical region, and hence the sphincters escape until late. Locally, evidences of spine disease appear early in the form of a kyphosis.

A *central* or *peri-ependymal myelitis* can rarely be recognized with

certainty. It produces less pain and irritation, but leads to muscular atrophy, disorders of sensations such as thermo-anæsthesia, disturbance of vasomotor and secretory nerves and visceral centres.

Pathology.—The pia mater is thickened over the affected region and often throughout the cord. The cord itself has a gray, discolored look at the affected level, and is usually shrunken or distorted and hard to the touch. In severe cases of secondary character it is reduced to a small size, and the membranes about it are thick and inflamed. In transverse myelitis a vertical area of only two or three inches is involved. The microscope shows that the prominent changes are loss of nerve structure, great increase of connective tissue, and increase in the number of vessels, which often have thickened walls. In the more seriously diseased part little is seen but connective tissue. In parts less diseased some nerve fibres are seen, many having evidences of partial disintegration. There is also a good deal of amorphous material studded with nuclei. Stellate cells, granule cells, and nerve cells in various stages of degeneration are present. In the parts less affected the signs of congestion and vascular irritation are more pronounced.

Diagnosis.—This must be made from progressive muscular atrophy and amyotrophic lateral sclerosis, pachymeningitis and spinal tumor; from locomotor ataxia, multiple sclerosis, and brain palsies.

In progressive muscular atrophy there is a peculiar atrophy without involvement of the sphincters or sensory disturbance. In pachymeningitis there is often a history of an injury; there is more pain in the back and a more marked anæsthesia. The sphincters are not involved. Pachymeningitis is also usually located in the cervical region. Tumors usually cause much more pain; the symptoms come on slowly and are more definitely localized. A spastic paraplegia occurs from brain disease and as a functional trouble. In either case there are no trophic or sensory troubles, nor is there involvement of the sphincters. In locomotor ataxia there is no great degree of motor paralysis, and there are peculiar ataxic and sensory disturbances. In multiple sclerosis there are eye symptoms, speech disturbances, and tremor. Paralysis from brain disease is almost always unilateral, painless, spastic, and free from disturbance of the visceral centres.

Prognosis.—Inflammatory processes have a tendency to cease when their reparatory and eliminative work is done. Chronic myelitis, however, is often, as has been stated, a destructive process due to some defect in vascular supply or to some mechanical irritation. Besides this, in the spinal cord secondary degenerations set in as soon as certain tracts are interfered with. Hence chronic

myelitis, after a period of improvement, generally progresses, and the prognosis is not very favorable. Still, patients may live from five to tweny-five years. Dorsal myelitis is the most favorable form; compression myelitis from caries can also often be successfully treated. Those forms which come on rather rapidly are more likely to cease progressing (Gowers). Serious involvement of the bladder is a bad sign, and naturally the prognosis is worse the more complete the paralysis.

Treatment.—In the earlier and progressive stage of chronic myelitis rest is imperative. The patient should lie down much of the time. Counter-irritation in the form of fly blisters, the cautery, or setons should be applied, and if no improvement results wet or dry cups used. The descending galvanic current along the spine should be tried; faradism and massage being used upon the limbs. Cold baths and cold applications must be prescribed carefully if at all. Lukewarm baths, 90° to 98° F., or half-baths with friction at 70° to 80° F., are more likely to be useful, but even these must be tried cautiously. The first baths should last not over five minutes and should be repeated only three or four times weekly. In later paraplegic and bedridden stages, electrical and hydro-therapeutic applications should be followed up patiently and persistently. The patient now may be allowed to remain and exercise in the lukewarm bath for some time.

Internally, iodide of potassium and mercury should be first given. After thorough trial with these remedies for six or eight weeks, the patient should be given courses of treatment with arsenic, nitrate of silver, phosphorus, and perhaps the chloride of gold. Pills of arsenite of sodium, gr. $\frac{1}{30}$, may be administered three or four times daily for two months; if benefit ensues, the remedy should be resumed after an intermission of three weeks. Phosphorus is best given in the form of an elixir in doses of gr. $\frac{1}{160}$ ter in die increased to gr. $\frac{1}{100}$. The remedy should be suspended for three days at the end of each ten days. Silver is given usually in the form of the nitrate (dose, gr. $\frac{1}{6}$ to $\frac{1}{3}$). Not more than one drachm should be given without a three-months' intermission. Some assert that the hypophosphite of silver and sodium, and the albuminate, are surer preparations. I do not advise the use of ergot. Strychnine in small doses is sometimes useful. For the bladder troubles, the internal use of boric acid, buchu, sandalwood, and similar drugs is useful. Mechanical and surgical measures may be of some help. In some cases suspension does good, but it may do harm. Tenotomy is justifiable for the purpose of straightening contractured limbs. In compression myelitis sus-

pension on an inclined plane and the plaster jacket or other support are indicated. Cases have been reported in which surgical operations for the relief of a supposed tumor have cured compression myelitis from Pott's disease. Sea voyages are often useful and are preferable to mountain climates. Rest, quiet, fresh air, and a very regular life are the essentials in all climates.

Acute Anterior Poliomyelitis (Infantile Spinal Paralysis, Acute Atrophic Paralysis).

Anterior poliomyelitis is a disease of the spinal cord characterized by a motor paralysis of rapid onset, followed by muscular wasting, without sensory symptoms. It occurs at all ages, but vastly oftener in infancy; hence it is often called infantile spinal palsy.

Etiology.—The average age at the time of attack is two years. Most cases occur under ten, and four-fifths of these occur under three. It may be congenital, *i.e.*, occur in intra-uterine life (Sinkler), and it may occur as late as sixty. Most adult cases occur under the age of thirty.* Rather more of the infantile cases occur in boys, and most of the adult cases occur in males. Race and climate afford no exemption so far as is known. The great majority of cases occur during the hot months of summer (Sinkler). Nearly eighty per cent (78.8) occur between June and September, inclusive. Heredity has an influence in only one or two per cent of cases.

Over-exercise and chilling of the body when heated are occasional causes. Infectious fevers (oftenest measles) precede the attack in about seven per cent (Sinkler). The disease has several times occurred as an epidemic. Dentition is rarely an exciting cause, as used to be supposed. Injuries and falls in a few instances appear to be the cause of the disease. The fact that the child is just beginning to walk at the period when most susceptible to poliomyelitis must be considered of importance, since the new movements call for an unusual activity of the spinal centres.

To sum up, *age*, *season*, and *infectious diseases* are the three most important etiological factors.

Symptoms.—There are rarely any premonitory symptoms. The

* Among 50 personal cases, 39 were in children. Of the latter there were 24 males, 15 females. Ages: Under six months, 1; half to one year, 8; one to two years, 10; two to three years, 5; three to four years, 3; four to six years, 3; six to eight years, 3. Final result was palsy, chiefly in right leg, in 14; chiefly in left leg, in 5; in both legs, 5; in right arm, 4; all four extremities, 1; the remainder in various combinations. Disease followed scarlatina in 1, pertussis in 2, cholera infantum in 1, some "fever" in nearly all cases.

patient is taken with a slight fever, 100° to 102° F., accompanied by vomiting, diarrhœa, or convulsions. In a few hours or a day paralysis develops; sometimes the paralysis is as much as a week in developing ("subacute form"). The fever lasts from one to three or four days. The paralysis rapidly reaches its height, then remains stationary for a time; then improvement sets in, which reaches a certain point and stops.

We have consequently:

1. A stage of invasion—a few hours or a week.
2. A stationary period—one to six weeks, usually two weeks.
3. A stage of improvement—six months to a year.
4. A chronic stage.

1. The stage of invasion may be so sudden as to suggest hemorrhage. Sometimes the child, after a restless, feverish night, wakes in the morning paralyzed. Usually the initiatory symptoms last less than a day. With or before the paralysis a fever of 100° to 102° F., vomiting, delirium, and, much less often, convulsions which are not severe, occur. After the general disturbance subsides there may be some pain in the back and limbs for a few days, and in rare cases the bladder is involved so that there is retention of urine. But the dominant symptom is motor paralysis.

The paralysis is oftenest paraplegia, next one leg, next the arms and legs, and after this various combinations. The eye muscles, laryngeal and respiratory muscles, always escape in infants. In older persons the facial nerve may be involved. In certain peculiar cases the cranial nerve nuclei are attacked, in association with the anterior horns. When the eye-muscle nuclei are involved it is called "polio-encephalitis superior;" when cranial nerve nuclei lower down are involved it is called "polio-encephalitis inferior."

2. The paralysis reaches its height in from one to four days. It remains at its height for from one to six weeks, and then improvement gradually sets in. In two or three weeks a wasting of the paralyzed limb may be noticed. It is flabby, its temperature lowered, and the reflexes are gone. Slight tenderness may be present, but there is no anæsthesia.

3. The stage of improvement lasts for from six to twelve months. There is gradual disappearance of the paralysis, beginning in the limbs least affected. This continues until the paralysis has left all but one or two limbs. As a rule, it is the legs alone that are finally left paralyzed. In a quarter of the cases both legs, and in half of the cases one leg, oftener the right, remain affected. The muscles waste and show the reaction of degeneration, viz., loss of faradic irritability, retention but lessening of galvanic irritability, sluggish

contractions, and sometimes polar changes. In the leg the anterior tibial group of muscles is oftenest affected, in the arm the deltoid and shoulder group. The atrophy, having reached a certain grade, finally ceases, and then a slight improvement may set in. After the end of a year not much further spontaneous improvement can be expected.

4. The temperature of the affected limb is lowered several degrees, the skin has a reddish-purplish, mottled look. The bones as well as muscles of the affected limbs do not grow so fast as those of the healthy limb. Hence in time the foot becomes smaller and the leg shorter. Owing to the contraction of unopposed muscles deformities occur. The most frequent are talipes equinus, talipes

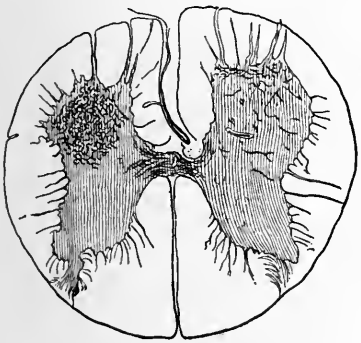


Fig. 119.

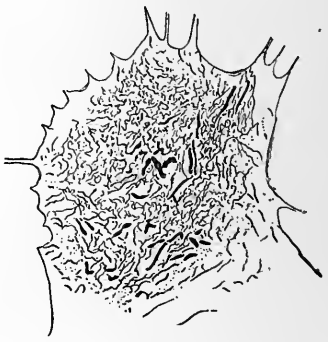


Fig. 120.

Fig. 119.—Anterior Poliomyelitis in Lumbar Segment (Demaschino).
Fig. 120.—Same, Much Enlarged, showing congestion.

valgus and varus. Deformities of the knees, contraction of the plantar fascia, and lateral curvature of the spine, also take place. The general health of the patient is usually good.

Pathology.—The disease is an acute exudative inflammation with destruction of tissue, but without suppuration. It affects chiefly the anterior cornua, especially of the lumbar and cervical enlargements (Figs. 119, 120). It is not, as a rule, diffuse, but often the brunt of the trouble is felt only by certain cell groups. These are destroyed, and after a time connective tissue takes their place. In rare cases the larger part of the central gray and some of the white matter are involved. Later a certain amount of sclerosis occurs in the lateral columns. The anterior roots and motor nerves atrophy. The muscle tissue also wastes, and its place is supplied by connective tissue.

Diagnosis.—The disease must be distinguished from multiple neuritis, spinal hemorrhage, cerebral palsies, birth palsies, and pro-

gressive muscular atrophy. The diagnosis can be easily made in almost all cases by remembering these facts:

1. The age of the patient.
2. The abrupt onset and rapid development of extreme paralysis.
3. The tendency to improve.
4. The absence of anæsthesia, bladder or rectal symptoms, rigidity, and pain.
5. The electrical reactions.
6. The arrest of growth of the limb. Multiple neuritis and progressive muscular atrophy rarely occur in children. Myelitis and hemorrhage are usually accompanied by sensory disorders, bedsores, and bladder troubles; cerebral palsies are usually unilateral and accompanied by symptoms of stiffness and exaggeration of reflexes.

Prognosis.—The patient rarely dies, either from the disease or its sequelæ. He always improves, but he hardly ever gets entirely well. The cases in which recovery is complete are those of simple exudative inflammation without any necrosis. Much can be done by careful and persistent treatment and by the help of orthopædic surgery, even in old cases. The usual course is for the patient to get back the use of all but one leg. He grows to adult life with this short and weakened member.

Treatment.—In the acute stage the child should be put to bed and kept there. Iodine or mustard plasters or leeches must be applied to the spine. Internally, a smart laxative and a diuretic must be given (calomel, gr. iij.; tartrat. potas., gr. xx.). Then tincture of aconite is to be administered in doses of one drop every half-hour as indicated by the fever. To this may be added sweet spirits of nitre. Rest is the most essential thing. The limbs should be kept quiet and warm. At the end of two weeks electrical applications may be very cautiously made to the limbs three times a week, if there is no tenderness or fever. After four weeks, electrical treatment should be given daily for a month, each limb being treated for only two or three minutes. After a rest of a fortnight another four-weeks' treatment may be given. Treatment should be thus applied intermittently till the end of a year. After this it can be continued or stopped according to the condition of the patient. In old cases daily treatment for one or two years will sometimes produce valuable results (G. M. Hammond). That form of electricity which causes muscular contractions most easily should be employed, and this is usually the galvanic current. Massage is a most important adjuvant to electricity. It is best given daily for not over ten minutes to a single limb. It is imperative also that the physician overcome any

contractures which develop, by splints, rubber muscles, and, if necessary, tenotomy. Warmth is very useful. The leg should be bandaged in cotton at night, and, if necessary, hot-water bottles placed beside it. Many parents cannot afford prolonged electrical

FIG. 121.—CASE OF CHRONIC ANTERIOR POLIOMYELITIS IN AN ADULT, showing the wasted hand and drop foot.

and massage treatment. In such cases they should be told to rub the limb twice daily with a stimulating liniment and wrap it in cotton or hot flannels at night.

The child should be taught to walk and exercise the limb as much as possible. Tricycles and gymnastic apparatus may often be brought into use.

Medicines are of little value in the chronic stage. Phosphorus, strychnine, iron, arsenic, cod-liver oil, physostigma, have been

recommended and are sometimes prescribed for the improvement of the general health and *solatii causa*.

Chronic Anterior Poliomyelitis.—This form of myelitis is very rare, and most careful examination must be made to exclude on the one hand multiple neuritis, and on the other progressive muscular atrophy.

Etiology.—Adults are chiefly affected, and men more often than women. Exposure, lead-poisoning, and syphilis are among the principal causes.

Symptoms.—The disease affects one or more of the extremities, often all four of them. There is a gradual paralysis, rapidly followed by atrophy, with degenerative electrical reactions. There is but little pain or other sensory disturbance. The sphincters are not affected.

The disease takes one of two courses:

1. After reaching its height, improvement gradually sets in and recovery may become nearly complete.

2. The disease steadily progresses until the patient presents the picture of a case of progressive muscular atrophy. In a few months, or at the most one or two years, death ensues. Occasionally, however, after reaching a very advanced stage, the process stops (Fig. 123) and a slight improvement may set in. These progressive cases of chronic poliomyelitis appear to stand half-way between ordinary chronic poliomyelitis and progressive muscular atrophy.*

Diagnosis.—The disease is distinguished from multiple neuritis by the absence of pain, tenderness, and anæsthesia; from progressive muscular atrophy by the rapid onset, the occurrence of paralysis first and wasting afterward, the early degenerative reactions, and the absence of fibrillary contractions. A history of lead poisoning may also help in the diagnosis.

The *treatment* is mainly symptomatic and must be carried out on the lines indicated under the head of Acute Anterior Poliomyelitis. Iodide of potassium, mercury, and strychnine should be given.

Acute and Chronic Senile Paraplegia.

A rather sudden paralysis of both legs sometimes attacks old people as the result of thrombosis or hemorrhage of spinal arteries. In these cases the symptoms come on suddenly, as in acute mye-

* *Subacute Spinal Paralysis of Duchenne.*—The diseases described under this head are chiefly cases of multiple neuritis. In a very few there are both neuritis and myelitis; in others the condition is one of minute focal spinal hemorrhages with secondary myelitis.

litis, only there is not much pain. The patient has sometimes a decided paralysis, and sometimes moderate paralysis but very great ataxia, depending on the arteries involved, whether postero-lateral or central. The patient usually improves rather rapidly and may regain a large part of his strength, but relapse is likely to occur. Rest and proper attention to diet and elimination are indicated.

A chronic form of paraplegia, slowly developing, is also seen in old people. It begins with simple weakness of the legs, followed by wasting and progressive development of a paraplegia. The sphincters eventually become involved. The disease affects the lower extremities first, but gradually extends, and finally involves the arms. The general characters are those of a progressive muscular atrophy; but the disease is distinguished from this by the fact that the sphincters become rather early involved, and that the paralysis and wasting go on together without any fibrillary contractions. The medulla and the facial and ocular muscles do not become involved. Clinically the disease cannot be distinguished from a chronic anterior poliomyelitis which takes upon itself a progressive type. On post-mortem, however, it is found that there is a softening of the gray matter in the anterior horns of the spinal cord, more marked in the lumbar swelling. This softening is apparently due to the thickening and obliteration of the blood-vessels from senile changes in them.

Dr. Gowers describes a disease which he calls senile paraplegia in which there are simple weakness of the legs and slowness of movement, without any atrophy, sensory disturbance, or alteration in the reflexes. He considers it to be a form of paralysis agitans. The disease which I have described, however, represents more truly a simple senile paraplegia. Very little can be done for this trouble therapeutically. The use of nitroglycerin, iodide of potassium, sparteine, digitalis, and general tonic and hygienic measures are indicated.

Acute Ascending Paralysis (Landry's Paralysis).

Acute ascending paralysis is a disease characterized by a rapidly developing paralysis which begins in the legs and then involves in turn the trunk, arms, respiratory and throat muscles, usually ending in death. There is little disturbance of sensation, no atrophy or changes in electrical irritability, and no involvement of the sphincters.

Etiology.—The disease is a rare one. It occurs chiefly between the ages of twenty and forty; men are affected oftener than women.

Exposure is an exciting cause, and it occurs sometimes after acute infectious fevers and syphilis. The form of rabies known as "paralytic" causes a disease which is apparently identical with Landry's paralysis.

Symptoms.—There may be slight premonitory symptoms for a few days, consisting of numbness in the extremities, pain in the back or limbs, and malaise. The first definite sign of the disease is weakness in the legs, which rapidly increases until in a day or two the patient cannot walk. The paralysis soon involves the arms and then the muscles of respiration; the medulla is last affected, and then respiration becomes difficult; swallowing and articulation may be impossible. In rare cases there are facial and eye palsies. During the course of the paralysis there is little pain or sensory disturbance, but some degree of anæsthesia may occur. The deep reflexes are abolished. There are no vasomotor and no secretory disturbances, no noticeable atrophy, and no degenerative reactions in the affected muscles. The bladder and rectum are involved only in rare cases. There may be slight initial fever, but none occurs after the disease has well set in. The mind remains clear.

The disease, as a rule, ends fatally, and it usually runs its course in less than a week. Death has occurred in forty-eight hours. On the other hand, death has been postponed three or four weeks.

In other cases the disease stops short of the medulla. The patient becomes totally or nearly paralyzed below the neck. He then begins slowly to improve, and this improvement continues for one or two years. Eventually a fair degree of health is obtained.

Variations.—The disease has been known to begin in the medulla or cervical region and descend.

Pathological Anatomy.—A number of different diseases have been described under the head of Landry's paralysis, and correspondingly a number of different anatomical changes have been found. Multiple neuritis, acute diffuse myelitis, and poliomyelitis existed in some cases. In others there was a dropsical exudation in the central canal of the spinal cord, or a hyaline change in the central arteries. In recent years decided changes in the anterior-horn cells have been detected by means of the Nissl stain; and it is pretty well decided that the disease is in its ordinary manifestations an acute toxæmia of the peripheral motor neuron, a fulgurating type of anterior poliomyelitis. It, however, affects in some cases the gray matter of the brain also. The paralysis is probably due to a poison of microbic origin. In some cases certainly this poison is that of rabies; but it is not impossible that other infections may pick out and suspend the functions of the anterior cornual cells, or,

as Gowers suggests, the "end brushes" of the motor tract which connect with these cells. This would explain the symptoms.

Sometimes the poison may be so great in amount and so irritating as to set up a myelitis or perhaps a neuritis. Cases illustrating these facts have been reported (Eichberg, Rosenheim, Putnam). But in most cases the patient dies before the toxin can produce any inflammatory reaction.

The prognosis is very grave, but not absolutely bad. If there is reason to suspect the case of being one of paralytic rabies, no hope can be offered.

Diagnosis.—This must be made from acute poliomyelitis, acute myelitis, and acute multiple neuritis.

Its acute ascending course, absence of fever, of anæsthesia, atrophy, decubitus, sphincter troubles, and especially the absence of degenerative electrical reactions are sufficient to enable one to make the diagnosis. The age of the patient, and the presence or absence of an alcoholic history should be considered.

Treatment.—This consists of warm baths or packs, counter-irritation to the spine, laxatives, and rest. Large doses of ergotin, gr. ij., every hour have been successful in one case. Salicylate or benzoate of sodium, iodide of potassium, and mercury may be tried.

CHAPTER XIV.

THE DEGENERATIVE DISEASES OF THE SPINAL CORD.

Introductory: The Nature and Types of Degeneration and Sclerosis.—The degenerative diseases of the spinal cord are sometimes called "system diseases," and some are often spoken of as scleroses. There are no true system diseases, however, except locomotor ataxia, progressive muscular atrophy, and amyotrophic lateral sclerosis. These should really be called "neuron" rather than "system" diseases, and this latter term is best not used as a basis of classification. The term "sclerosis," also, is somewhat misleading. Properly speaking it is the fibroid (and neuroglia) induration which results from degeneration, destruction, or inflammation of nerve tissue. We speak of *degenerative* sclerosis, of an *inflammatory* and of a *neuroglia* sclerosis, or of a sclerosis of mixed origin, according to the nature of the primary disease which caused it. The words "degeneration" and "sclerosis" are often used to indicate the same thing, one being the pathological name, the other the anatomical. I shall use the term "sclerosis" here in its pathological sense, meaning the process of hardening, in presenting a classification of the degenerations of the spinal cord.

- Spinal scleroses or degenerations.
  - *a.* Primary and degenerative.
    - Posterior spinal sclerosis (locomotor ataxia).
    - Lateral sclerosis.
    - Combined sclerosis.
    - Progressive muscular atrophy, amyotrophic lateral sclerosis.
  - *b.* Secondary.
    - Ascending and descending degenerations.
    - Chronic myelitis and sclerosis following destruction of cord.
    - Multiple sclerosis.

A few words of explanation are needed regarding these different conditions, since a world of confusion has been made on account of the different standpoints taken by writers when nerve pathology was young.

a. Primary degeneration or primary sclerosis, as one may say

for convenience, is a process which begins in the nerve tissue itself and ends in its atrophy, with substitution of connective tissue. As to its *nature*, so far as the microscope shows us, it is a gradual decay and death of the neurons. In some sclerotic processes, like locomotor ataxia, this decay is accompanied by the development of irritating products, leucomains or toxalbumins, which may produce so active a change in the connective tissue as to lead to something resembling a secondary or reactive inflammation. This is

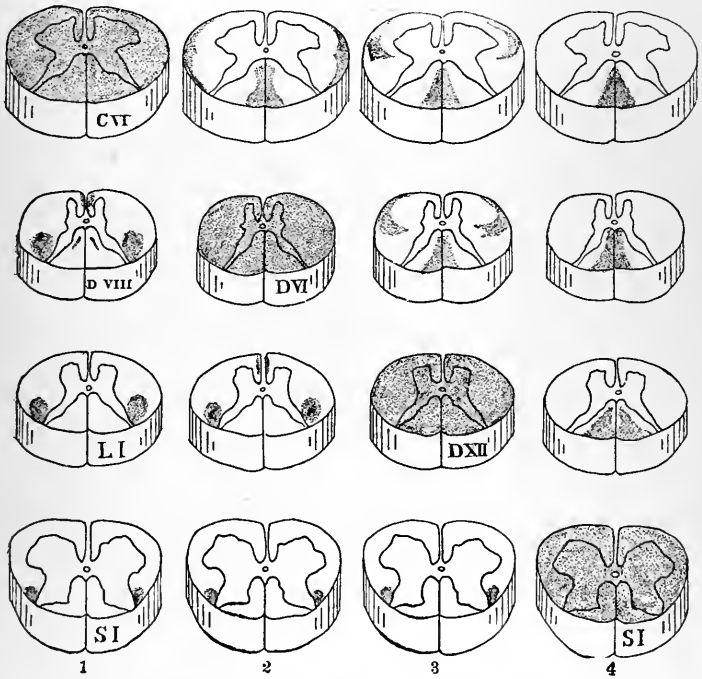


FIG. 122.—SHOWING THE TRACTS AFFECTED IN SECONDARY DEGENERATIONS OF THE SPINAL CORD IN LESIONS AT DIFFERENT LEVELS. 1, Descending degeneration after lesion at sixth cervical; 2, ascending and descending degeneration, lesion at sixth dorsal; 3, ascending and descending degeneration, lesion at twelfth dorsal; 4, ascending degeneration, lesion at first sacral.

never of high grade, however, and in some forms of tabes is very slight.

In progressive muscular atrophy the decay and death produce few irritating products, though enough, perhaps, to account for the fibrillary twitchings and occasional hypertonic condition of the muscles.

The ultimate cause of these degenerative processes is not known. The progressive character of the diseases like locomotor ataxia and

progressive muscular atrophy would lead one to think that there is a poison at work and constantly acting on the diseased tissue. So far, all bacteriological examinations have failed to disclose any microbe, but the fact that many degenerative processes follow infectious fevers or syphilis has led to the suggestion that pathogenic germs have poured into the system a poison, or have so modified the cellular nutrition that there is a poison constantly thrown out, which irritates and destroys certain areas of nerve tissue. The normal immunity of certain neurons to metabolic poisons is lost.

All the primary degenerations or scleroses have a certain degree of kinship. Their causes are in many respects the same, the course of all is uniformly progressive, and one not very infrequently complicates another. The sharpest distinctions are found between those affecting the peripheral motor neurons and the peripheral sensory neurons. Degenerative processes implicating the former are much rarer and their course is more rapid and fatal.

The degenerations of the spinal ganglia and the peripheral sensory neurons are more common, slower in course, different in etiology, and much more varied in symptomatology. Locomotor and the hereditary ataxias furnish examples of this type.

b. Secondary Degenerations of the Spinal Cord.—When any of the long-fibre tracts of the cord are cut across or destroyed, there soon results a degeneration. This extends up or down in accordance with the direction in which the tracts carry impulses. Thus, when the crossed pyramidal tract is cut across the degeneration extends down; when the column of Goll is involved it extends up. The degenerative process begins almost immediately and is complete in a few weeks. The myelin sheath swells, gradually breaks up, and disintegrates; the axis cylinder is involved next. At the same time the connective tissue proliferates and takes the place of the wasted nerves. Finally, long tracts of connective tissue have taken the place of the nerve tissue. The process may not be a complete one if the lesion does not entirely destroy the tract. The short-fibre tracts degenerate only a little way up and down.

Secondary degenerations complicate and add to the pathological change in all organic diseases of the cord. In brain disease, involving the motor tract, as in hemiplegia, secondary degeneration extends into the cord and adds to the seriousness of the disease. Degenerations of the spinal cord, however, do not extend up to the brain except in the case of disease of the cerebellar tracts.

Those forms of sclerosis found in chronic myelitis are similar to the connective-tissue scars following destructive inflammation else-

where. A person who has a chronic myelitis has a cicatrix in his spinal cord. The sclerosis of multiple sclerosis is probably inflammatory also, but it is a neuroglia rather than a connective-tissue cicatrix.

The diagrams in Fig. 122 show the principal secondary degenerations. Thus a lesion at the level of the sixth cervical segment shows a descending degeneration in the pyramidal tracts and the comma-shaped tract of Schultze below. A lesion at the sixth dorsal segment shows ascending degeneration in the column of Goll,

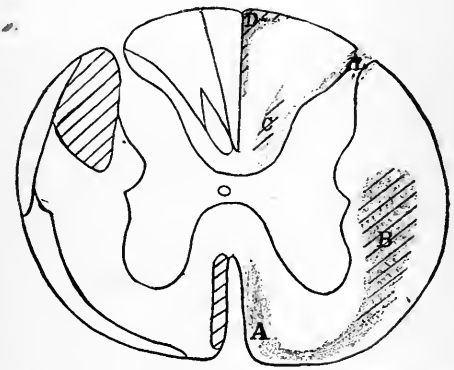


FIG. 123.—SHOWING ON THE RIGHT SIDE THE MINOR AND SHORT TRACTS DEGENERATING AFTER SECTION OF THE CORD. *A*, ascending sulco-marginal; *B*, intermediate lateral (descending); *C*, comma tract (descending); *D*, posterior sulco-marginal; *L*, Lissauer's. Those tracts lined across degenerate downward, the others upward.

direct cerebellar tracts, and antero-lateral ascending tracts above, and in the pyramidal tracts below.

Short Degenerating Tracts.—By the use of more delicate stains other degenerating tracts have been discovered.

In the lateral column, scattered fibres exist which degenerate downward. This is the "intermediate tract of the lateral column" (Fig. 123, *B*). Others of the same kind are found in the anterior column. This is the "sulco-marginal tract" (Fig. 123, *A*). The descending fibres, some of which degenerate downward, some upward, come in part from the cerebellum, forming a descending cerebellar tract. In part they are long commissural fibres connecting upper and lower spinal segments.

In the posterior column are two small tracts called the "comma tracts," because of their shape. They lie at the junction of the columns of Goll and Burdach in the position shown in Fig. 123, *C*. The fibres degenerate downward in transverse lesions of the cervical and dorsal cords. Usually this does not extend more than an inch. The tract may escape degeneration in locomotor ataxia

There is a descending tract also in the lumbo-sacral region of the cord, corresponding in a measure to the oval zone of Flechsig. As described by Bruce, it lies along the posterior median septum above, while below it spreads out along the posterior margin. Bruce has called it the posterior septo-marginal tract (Fig. 125, *D*).

LOCOMOTOR ATAXIA (Posterior Spinal Sclerosis, Tabes Dorsalis).

Definition.—Locomotor ataxia is a chronic progressive disease, involving primarily the posterior spinal ganglia or analogous neurons, and later the spinal cord and peripheral nerves. It is characterized clinically by inco-ordination, pains, anæsthesia, and various visceral, trophic, and other symptoms, and anatomically by a degenerative sclerosis chiefly marked in the posterior columns of the cord and posterior roots, and to a less extent in the peripheral nerves.

Forms.—Besides the common and typical form, there are anomalous and complicated types.

Types.	1. Common form. 2. Neuralgic. 3. Paralytic. 4. With initial optic atrophy.
Complicated forms.	With muscular atrophy. With other scleroses. With general paralysis.

Etiology.—The disease occurs oftenest in middle life, between thirty and forty, next between forty and fifty. It may occur as early as the tenth and as late as the sixtieth year. In the very early cases it is usually due to hereditary syphilis. It is much more common in males. Hereditary influence is very unimportant and is only indirect, *i.e.*, the parents may be neurotic. Diathetic influence is slight. Exposures to wet and cold, combined with muscular exertions, are effective causes. Soldiers, travellers, and drivers are rather more susceptible. Excessive railroad travelling, excessive dancing with exposure, favor the development of the disease. Excessive sexual intercourse, combined with irregular living, is a predisposing cause. Syphilis is by far the most important single factor. A history of the disease is obtained in from sixty per cent to ninety per cent of the cases. In my own cases sixty-five per cent had had syphilis. The patient has usually contracted the venereal disease ten or fifteen years before, and has rarely had noticeable secondary symptoms.* Syphilis is not a direct factor,

* The following statistics from my own experience show the physiognomy of the disease in a cosmopolitan American city. Total cases, 190; males, 173;

but prepares the system for the degenerative process. Syphilis followed later by excesses—mental or physical—and by exposures especially tends to produce locomotor ataxia. Lack of proper treatment for syphilis is believed to favor the development of the disease, but a careful study of the statistics of my clinic and of his own cases by Dr. Joseph Collins seems to show that antisyphilitic treatment as at present carried out does not prevent the disease if the other favoring conditions, such as exhausting work and sexual or alcoholic excesses, are present.

Among other causes are profoundly depressing emotions, acute infective diseases like typhus, pneumonia, and rheumatism, difficult labors with severe hemorrhage, prolonged lactation, injuries with shock, and excessive smoking.

Locomotor ataxia in a somewhat atypical form may result secondarily from gummatous inflammation of the spinal meninges, from a tumor, and possibly from an ascending neuritis.

Symptoms.—The disease is generally divided into three stages: the initial or pre-ataxic, the ataxic, and the paralytic.

1. The initial stage. The patient first notices a slight uncertainty in walking, especially at night; he has numb feelings in his feet, and at times darting pains in the legs or rectum. His sexual function becomes weak, his control over the bladder slightly impaired. He has temporary attacks of vertigo and of double vision. A continuous sense of profound weariness oppresses him, even though he has made no exertion. The knee jerk is lost. Such symptoms may last a few months or several years.

2. The ataxic stage. The gait now becomes so unsteady that others notice it; the patient has to use a cane, and when walking watch his feet and the ground. If he stands with his eyes closed, he totters and may fall. His feet feel as though there was a layer

females, 17. Ages when disease began: Twenty-one to thirty, 19; thirty-one to forty, 84; forty-one to fifty, 60; fifty-one to sixty, 22; sixty-one to seventy, 5. Average age at time of onset, 40; beginning a year or two earlier in private patients, and in those with a history of syphilis and active antisyphilitic treatment (Collins). Percentage of syphilis, 60. This is making the most liberal allowances. In one set of eighteen hospital patients, all carefully investigated, the percentage was 70. Average period between infection and tabes, fifteen years, ranging from one and one-half to twenty-five years. This is much longer than Erb's estimate, but is reached by three independent studies of my cases. Average duration of disease when seen by me, eight years; average duration of life in five fatal cases, twelve years, ranging from five to twenty years. Complications: optic atrophy, 6 per cent; marked arthropathies, 5 per cent; with general paresis, 4 per cent; paraplegia, 1.5 per cent; hemiplegia, 1.6 per cent; eye palsies, 8 per cent.

of cloth or cotton between the soles and the ground. Paroxysms of lightning-like pains attack the legs, and tests show anæsthesia present in the toes and feet or in patches on the legs. A sense of constriction is felt around the waist. The sexual power is often lost; the bladder is weak, and care has to be taken to empty it. The bowels are constipated; at times he has attacks of intense pain in the epigastrium, with vomiting and perhaps a diarrhœa coming on without cause. The pupils are small and do not react to light but do react to accommodation; vision is still good. The inco-ordination and pain and anæsthesia after a time begin to affect slightly the arms. This stage lasts several years.

3. The paralytic stage. After several years with various remissions and improvements, the patient loses altogether the power of walking. His legs are somewhat wasted, but the muscular strength is fairly good. The anæsthesia and ataxia are very great. The patient does not feel the prick of a pin or touch of the hand; nor with closed eyes does he know where his legs are. His bladder is anæsthetic and paretic, so that the urine has to be drawn. The pains are much less, but are still present at times. The arms are more involved, but never so seriously as to make them useless like the legs. The intelligence remains good, and the patient may continue bedridden for years, dying finally from some intercurrent affection.

The following table shows the prominent symptoms in the usual order of their appearance:

	First Stage. (Half to twenty years.)	Second Stage. (Two to ten years.)	Third Stage. (Two to ten years.)
Motor	Eye palsies. Ataxia. Muscular weakness.	Less. Increased. Paresis.	Increased. Paraplegia.
Sensory	Pains.	Pains. Anæsthesia.	Pain less. Increased.
Excito-reflex	Loss of knee jerk. A.-R. pupil.		
Trophic	Arthropathies.	More rare.	Rare.
Visceral	Sexual weakness. Vesical weakness. Constipation.	Increased.	Increased.
Special senses	Diplopia. Optic atrophy.	Rare. Rare. Deafness.	Increased. Increased. Paralysis of accommodation.

The symptoms must now be analyzed more closely.

Locomotor and *static ataxia* are present very early, but only to a moderate extent. Tests, such as making the patient walk and stand with the eyes closed, noting the position of limbs and the weight of objects, will reveal an ataxia due largely to beginning anæsthesia of the joints and tendons. By the use of the ataxigraph, one can with care assure himself that the patient has an excessive degree of static ataxia. In my experience, when the ataxigraph records over three inches' oscillation, the patient not being paraplegic or under the influence of any drug, it is abnormal.

The patella-tendon reflex or knee jerk is abolished very early in all typical cases. This constitutes a very important symptom, therefore.

The gait and station in ataxia are characteristic. In walking, the patient keeps his eyes on the ground and on his feet. The latter he throws out rather forcibly, owing to overaction of the extensors of the foot. In watching such a patient walk barefooted, the extensor tendons can be seen to stand out with each forward movement of the limb. The foot is brought down sharply on the heel and the legs are spread apart a little. Turning a corner, turning around, and going downstairs are done awkwardly, and the patient is apt to totter and fall. Walking on a chalked line is very difficult; so also is walking backward. The gait improves after the patient walks a while, and he will generally say that the practice of walking does him good. Still, he soon gets tired (Fig. 124).

FIG. 124.—SHOWING STATION IN SECOND STAGE OF LOCOMOTOR ATAXIA.

Severe rectal *neuralgia*, associated perhaps with hemorrhoids, is sometimes an early symptom. Persistent neuralgia and functional disturbance of the bladder and rectum should cause suspicion of ataxia. Lancinating or lightning pains occur and are very characteristic. The pains dart down the legs along the course of the sciatic, or they suddenly appear as patches of pain on the foot or leg or thigh ("spot pains"). The pain comes unexpectedly and with such severity that the patient involuntarily jumps or jerks the limb.

He speaks of his "jerking" and "twitching pains." The pains may affect the bowels or be felt as a squeezing sensation around the waist (girdle pains). The pains of ataxia are often the most obstinate and distressing symptom. They usually come on in great intensity once or twice a month, and last for two or three days. They then leave the patient for a time. They are often worse in cold and damp weather. In some cases the pains are almost continuous,

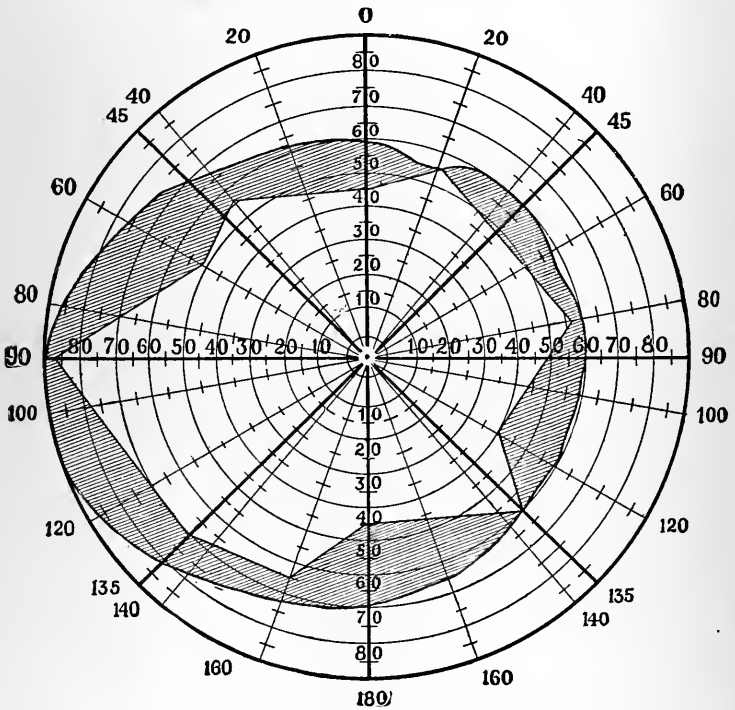


FIG. 125.—IRREGULARLY CONTRACTED VISUAL FIELD IN CASE OF TABES WITH OPTIC ATROPHY, LEFT EYE (Berger).

coming on, if not every day, at least two or three times a week. Such cases are associated with much cutaneous hyperæsthesia, especially during the attacks. This type of cases is called "*the neuralgic.*" The patients rarely have as much ataxia, paresis, or visceral troubles as the typical forms present, and in certain respects such cases are favorable.

The pains of the disease continue well into the second and even third stage. Meanwhile the anæsthesia becomes much more marked. It affects most the feet and next the legs, rarely extending much over the thighs, but passing to the fingers and hands. The anæs-

thesia is greatest to pain, but touch and temperature sense are also involved. There is often delayed conduction and polyæsthesia; many other curious perversions of the cutaneous sense are noted. Some anæsthesia usually develops over the finger tips and hands, and sometimes a band of anæsthesia develops about the trunk. The facial and cranial nerves are not much affected, but there may be trigeminal neuralgia.

Optic atrophy occurs in about six per cent of cases, in my experience. Optic atrophy usually develops in the pre-ataxic stage. If a patient has reached the *second stage without it, he will probably escape it altogether.* Cases with ocular paralyses are slightly more disposed to it (Berger). It attacks the left eye oftener than the right. The atrophy begins sometimes with increased sensibility to light, flashes of light, and muscæ volitantes. With the failing vision, disturbance of color sense often and contraction of the visual field always occur. This contraction is irregular, with sector-formed defects; not hemiopic (Fig. 125). The atrophy progresses slowly with slight remissions. It may cease its progress, but this is rare. Blindness comes in about three years. Ophthalmoscopically there may be seen slight evidence of congestion in the early stage; later, pallor of the disc, which finally becomes grayish.

Disorders of *hearing* are frequent in tabes, occurring in about one-fourth of the cases, but in the majority of instances the aural trouble is an accidental complication due to middle-ear disease. Primary atrophy of the auditory nerve is very rare, as might be expected, since this nerve is structurally not like the optic nerve. Its existence has been inferred on clinical grounds. Another form of tabetic deafness is of trophic origin and due to a sclerotic condition of the middle ear (Treitel). It is caused by involvement of the trophic or vasomotor fibres of the fifth nerve.

The senses of *taste* and *smell* are rarely affected.

The *eye muscles* are implicated in some way in nearly all cases of tabes. The following are the disorders:

1. Loss of the light reflex, and myosis. 2. Sympathetic-nerve ptosis. 3. Paralysis of branches of the third nerve. 4. Paralysis of the sixth nerve.

Paralyses of the ocular muscles (third and sixth) occur rather oftener in distinctly syphilitic cases. Other ocular troubles are not influenced by exudative syphilis. Ocular palsies are early symptoms of the disease, occurring, as a rule, in the pre-ataxic stage.

1. Loss of light reflex and pupillary rigidity. The pupils are small and sometimes uneven; they do not respond to light, but they

do to accommodation. This condition is known as the *Argyll-Robertson* pupil. In early stages the light reflex may be simply sluggish. In the late stages the accommodation reflex is also lost. The Argyll-Robertson pupil is practically found only in tabes and in general paresis. The ocular skin reflex usually disappears early. The myosis in tabes is due to paralysis of the sympathetic dilating fibres. The pupils are sometimes irregular in shape. In some cases there is a loss of both light and accommodation reflex. This is especially characteristic of an exudative brain syphilis (Sachs).

2. Sympathetic-nerve ptosis. A slight drooping of one or both lids is not infre-

FIG. 126.—ARTHROPATHY OF ANKLE.

FIG. 127.—ARTHROPATHY INVOLVING KNEES AND LONG BONES OF LEGS.

quent. It begins early and progresses slightly up to the later stages of the disease. It is due to paralysis of the sympathetic-nerve fibres of the lid.

3, 4. Paralysis of the external eye muscles. The external rectus is oftenest affected of single muscles, but the various branches of the third nerve taken together are oftener involved than the sixth. Of the third nerve's branches, the levator palpebræ and internal recti muscles are oftenest involved. There may be multiple palsies. These occur oftener in syphilitic cases. Progressive ophthalmoplegia may be associated with tabes. The ocular nerve palsies may be transitory or permanent. Those occurring in the pre-ataxic stage

are usually transitory, lasting a few hours, days, or weeks. Cases have even lasted two years and got well. The permanent palsies develop usually in the later stages. The early palsies are usually due to a syphilitic exudation at the base of the brain; the late pal-

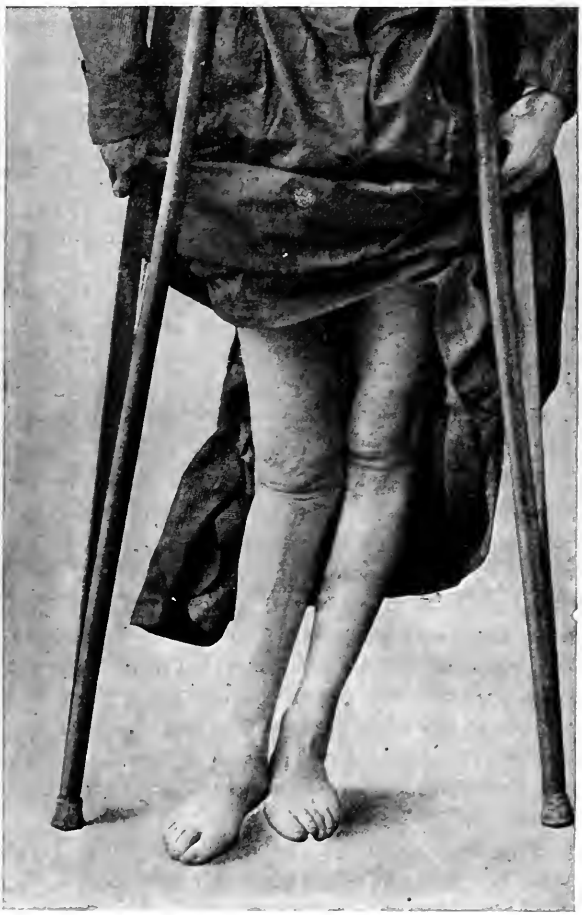


FIG. 128.—ARTHROPATHY OF KNEES IN TABES, SHOWING RELAXED JOINTS.

sies are usually due to degenerative lesions of the nuclei of the ocular nerves.

The *arthropathies of locomotor ataxia.* Degenerative diseases of the joints, technically known as arthropathies, and spontaneous fractures of bones form important symptoms of tabes. They occur in ten per cent (Charcot) or five per cent (author) of cases.

The arthropathies are three or four times more frequent than

the fractures. The joints oftenest affected are the knees, ankles, and hips; but the elbow, shoulder, wrist, and small joints may be attacked.

Spontaneous fractures occur oftenest in the shaft and neck of the femur, next in the legs, forearm, humerus, and clavicle. The pelvis, scapula, vertebræ, and under jaw may be fractured. Arthropathies are often accompanied by fractures, especially of the heads of the bones. The two sides of the body are about equally affected.

The arthropathies are characterized by a sudden, apparently spontaneous painless swelling of the joint. The symptoms may develop in twenty-four or forty-eight hours. In rare cases there is a history of some preceding rheumatic pains or of an injury. After a time there is an osseous hyperplasia of the joint, which becomes enlarged to enormous proportions. There is also a tendency to luxation of the joint. It crepitates on moving. There is no tenderness on pressure; the hand finds evidence of synovial exudation, roughened surfaces, and perhaps fractures of the enlarged parts. In the milder forms there are simply swelling from synovial exudation and some enlargement of the bones with roughened surfaces. After a few weeks this swelling may subside and the joint return to nearly its natural size. In other cases the process progresses, the ligaments relax, the bones of the joint can be moved about freely, and luxations are easily produced. There is still no pain, but the limb becomes almost or entirely useless on account of the loose and relaxed condition of the parts (Fig. 130). As time goes on, some absorption takes place and the head of the bone may almost disappear. The arthropathies have been divided into benign and malignant, but no sharp line can be drawn or certain prognosis made in the early stage. The arthropathies appear in the prodromal and early stage of the disease in over half the cases, and are often at first unrecognized. One-third occur after the tenth year of the disease.

The spontaneous fractures are usually brought on by a slight trauma, such as a fall. Violent muscular movements may produce them. They also are painless, as a rule. The fractures usually heal well, often with abnormal readiness, but occasionally there is delay, and often healing is accompanied by great throwing out of callus.

Pathologically the arthropathy is a rarefying osteitis. It does not differ anatomically from arthritis deformans, except that fractures may accompany it. Clinically the chief difference lies in the abruptness, spontaneity, and painlessness of the process. The disease, on the whole, cannot be considered specifically different from arthritis deformans, modified by the analgesia of the parts. It is

due probably to a degenerative change in the nerves supplying the joints and bones. The process may begin in the cartilage, bone, or ligaments. Eventually all these parts are involved. There is congestion of the synovial membranes with hydrarthrosis, then atrophy and rarefying hypertrophy of the epiphyses, relaxation of the ligaments, formation of osteophytes and bony stalactites. There may be a rarefying osteitis of the long bones, without much joint involvement at first (Fig. 127).

Various *trophic disturbances* of the skin may appear, generally late in the disease. The most common are herpes and lichen. Besides these, bullæ, transitory erythema, urticaria, eczema, pemphigus, ecthyma, ulcers, ichthoysis, and petechiæ have been described; but they are rare and often only accidental complications. A peculiar round perforating ulcer sometimes develops on the sole of the foot, often as the result of cutting a corn. In rare cases the nails and teeth fall out. In distinctly syphilitic cases there is usually baldness.

FIG. 129.—PERFORATING ULCER OF FOOT IN TABES.

Peculiar "crises" of various kinds occur in tabes. The most common are *gastric crises*. These consist of attacks of intense pain extending from the groin to the epigastrium or encircling the waist, accompanied by vomiting and sometimes diarrhœa. The attacks are often associated with pains in the legs. They last two or three days, then pass away.

Laryngeal crises consist of attacks of spasm of the adductors or paralysis of abductors, with noisy, croupy respiration. The attacks come on suddenly, the patient coughs and struggles for breath, and he may be seized with vertigo and fall down. The pulse may be very fast. The paroxysm lasts for a few minutes to several hours. The symptoms are very distressing, but not dangerous. Paroxysms of cough have been described as "bronchial crises." There are also *cardiac crises*, in which there are dyspnœa and rapid heart beat and sense of suffocation resembling angina. The heart itself sometimes is diseased, but whether from neurotrophic disturbance or not is doubtful. The pulse is often small, rather rapid, and weak. The laryngeal and heart crises both depend on a degenerative disease

and irritation of the vagus, and may be more or less united in symptoms.

A *sense of great weariness* and heaviness in the limbs, present constantly, no matter how much rest is taken, is a characteristic early symptom, and is due to an irritability of the nerves of muscular sensibility.

Muscular atrophies occur sometimes in tabes. They are of three kinds: 1st, a true progressive muscular atrophy due to degeneration of trophic and motor cells; 2d, localized muscular atrophies due to degenerative atrophy of nerves; 3d, a general wasting. Under the first head one finds ophthalmoplegia, bulbar paralysis, and spinal amyotrophy: Under the second, wasting of certain groups of muscles in the legs or arms.

Besides these, there is a generalized atrophy which occurs in the paralytic stage and is due probably to a slight involvement of the anterior horns in the progressive process that affects the cord.

Attacks of hemiplegia in rare instances occur in tabes. They are usually of temporary character and occur early in the disease. They may come on late and are then more likely due to acute softening. In all cases the trouble is due to embolism or to disease of the cerebral vessels, of syphilitic origin.

Acute paraplegia comes on occasionally also, and this sometimes almost disappears.

The *sexual power* may be at first greatly exaggerated; but this is rare, and usually there is progressive weakness and loss of desire. The bladder and sexual functions are rarely entirely lost and rarely equally impaired in the first stage; one may continue good while the other is affected moderately. Usually the sexual function goes first.

Some *cerebral symptoms* occur in tabes, chiefly in the early stage. They are insomnia, which may be very obstinate; and occasional vertigo. An irritability of temper and tendency to despondency, sometimes noted, cannot be considered unnatural. Apoplectiform and epileptiform attacks are described, but are very rare, and should cause a suspicion of a complication. The disease in very rare cases terminates in general paresis.

Course.—The disease has been termed progressive, but it is not so in a large number of cases. With proper treatment the symptoms can often be kept in control for years. The first stage may last twenty years or more; the second stage five to fifteen years. The total duration of the disease varies enormously, ranging between three and thirty years. A few acute cases have been observed, running a course of less than a year.

Complications.—These are acute myelitis, generally syphilitic;

lateral sclerosis, progressive muscular atrophy, hemiplegia from embolism or endarteritis, general paresis, and heart disease.

Pathological Anatomy and Pathology. — The characteristic changes are found in the spinal cord, posterior spinal ganglia and posterior roots, and to a less extent in the peripheral nerves. The spinal cord usually is reduced in size and flattened antero-posteriorly; the pia mater is thickened somewhat. One can see with the naked eye that the posterior columns of the cord are shrunken and have a grayish appearance.

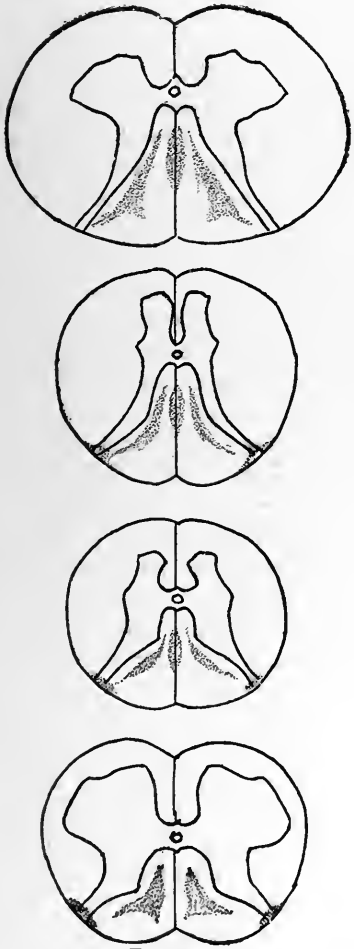


Fig. 130.

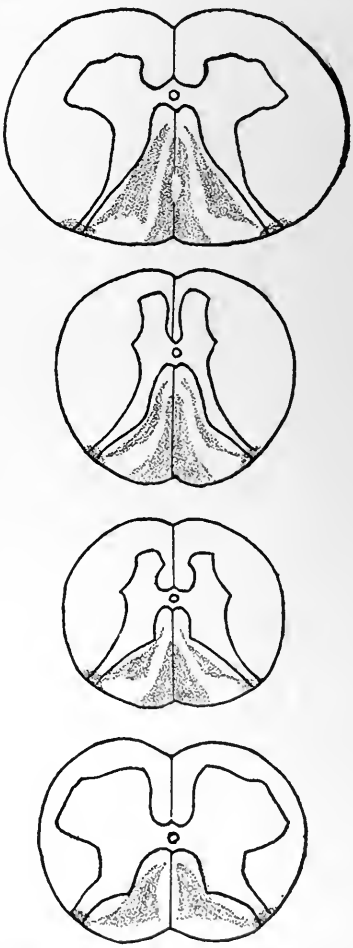


Fig. 131.

Fig. 130.—Locomotor Ataxia, showing areas affected in first stage at five different levels. Drawn from specimens in author's possession and from comparative study of over thirty other figures.

Fig. 131.—Locomotor Ataxia, Second Stage.

Under the microscope it can be seen that the white matter of the posterior columns is very seriously affected; the nerve tisue has disappeared, and its place is taken by connective and neuroglia tissue through which a few nerve fibres still run. The walls of the blood-vessels are somewhat thickened, but not remarkably so, nor is there any notable evidence of congestion or excessive vascular irritation.

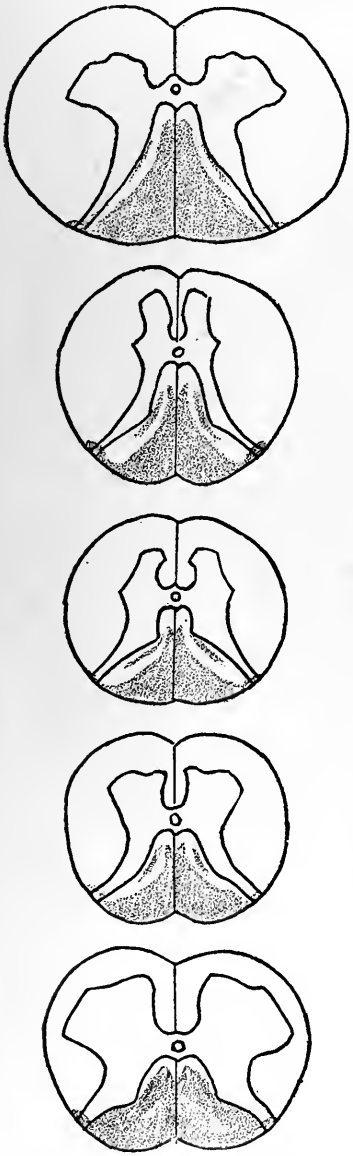


Fig. 132.—Locomotor Ataxia, Last Stage.

The part of the posterior column first affected is a vertical streak lying in the middle root zone between the posterior median (columns of Goll) and posterior external columns (columns of Burdach) (Fig. 130). The segments first and most affected are those of the upper lumbar and lower dorsal region. Besides this area the rim zone or column of Lissauer is also early involved. As the disease progresses it extends upward and spreads laterally so that finally all of the posterior column is changed into a dense connective-tissue mass through which only a few nerve fibres run. The part last and least involved is that lying just posterior to the commissure (anterior root zone of Flechsig, ventral fundamental column) and that lying just mesial of the posterior horns (external part of the middle root zone) (Fig. 132). There is sometimes a degeneration of the antero-lateral ascending tract (Gowers' tract), and very rarely of the cerebellar tract. The pyramidal tracts are involved only in complicated cases. The cells and fibres of the column of Clark are often involved in advanced cases (Fig. 133). The gray matter of both the posterior and anterior

horns may show some degenerative changes, viz., decrease of the fibre network and atrophy of the cellular elements.

The posterior roots are usually involved, the process extending as far as the spinal ganglia, which also show some degeneration (Figs. 134, 135), but the lesion is not strikingly marked here in all cases, and sometimes the spinal ganglia are nearly healthy, although the posterior columns are diseased. The anterior roots are normal.

The process begins in the upper lumbar cord; the sacral cord is usually much less affected. However, the exact initial point of attack varies, and this accounts for the variation in the symptoms. Cases that begin with decided bladder and genital symptoms probably start low down; cases which go for a good while with only ataxia, loss of knee jerk, and pains begin higher; while in the brachial or arm-type cases the process begins in the cervical enlargement.

The peripheral nerves are diseased in a large number of the advanced cases. The nerves of the leg are most involved. The process is a degenerative atrophy or neuritis (Figs. 136, 137). It affects the extremities of the nerves first and slowly extends upward, seldom reaching the large trunks.

Sometimes the disease begins in the optic nerves or possibly in the bipolar visual cells of the retina. This

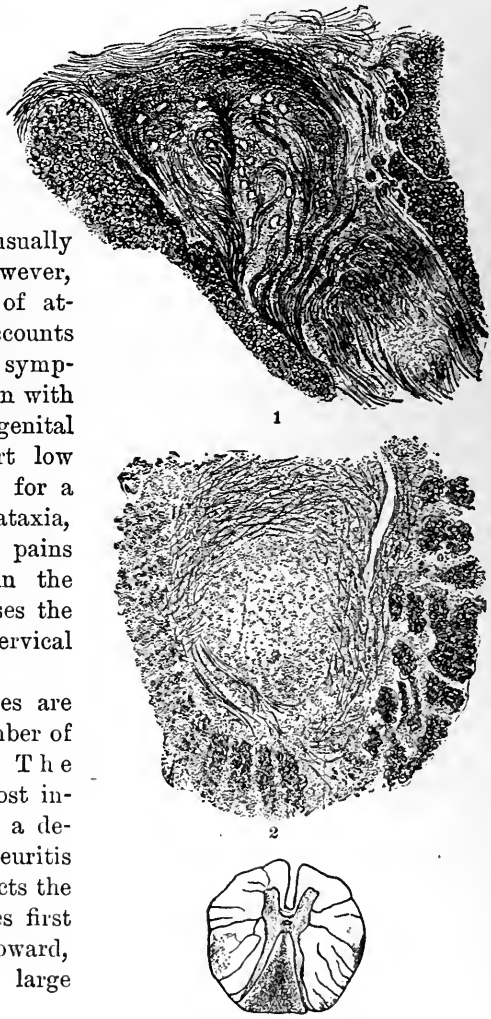


FIG. 133.—SHOWING COLUMNS OF CLARK. 1, Normal; 2, diseased, in case of tabes in third stage; with degeneration of posterior columns and cerebellar tracts as shown in 3 (Oppenheim).

should be the case in order to observe the neuronic homologies. The process here is an atrophy beginning at the periphery and extending brainward. The third, fifth, and sixth nerves are occasionally involved; still more rarely the olfactory and auditory. The vagus nerve and sometimes its nucleus and that of the glosso-pharyngeal are implicated, it may be, rather early in the disease. It is believed that these facts explain many of the laryngeal and visceral crises.

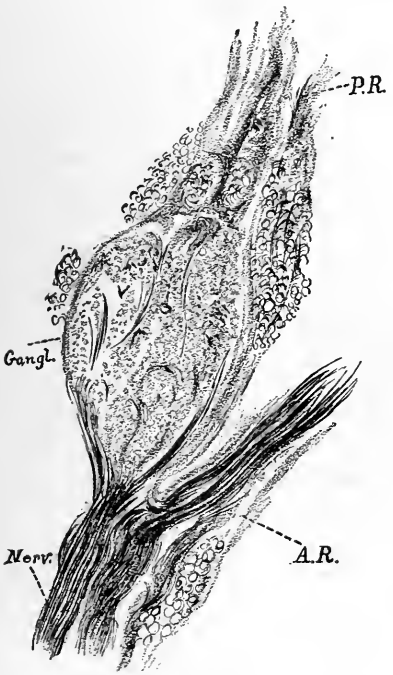


Fig. 134.

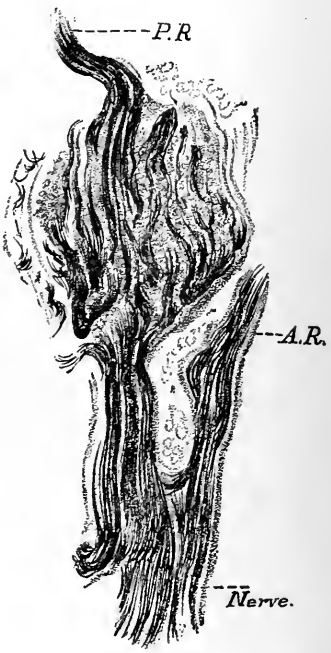


Fig. 135.

Fig. 134.—Posterior Spinal Ganglion in Third Stage of Tabes. *P.R.*, posterior root; *A.R.*, anterior root (Oppenheim).

Fig. 135.—Healthy Spinal Ganglion.

Pathology.—The pathology of locomotor ataxia cannot be thoroughly understood without a knowledge of the pathology of syphilis in its relation to the nervous system—a subject which is discussed later. It may be said here, however, that syphilis leads to two sets of changes in the nervous system: one, the earlier, is inflammatory; the other, and later, is degenerative. The inflammatory changes attack the blood-vessels and serous membranes, leading to the deposit of exudates, and these are the characteristics of secondary syphilis when it involves the nerve centres. The degenerative changes attack the nerve tissue directly. They follow long after the infec-

tion and are the result of the syphilitic poison which has been permeating the system. Degenerative syphilis of the nervous system is not, strictly speaking, syphilis at all, but rather the effects of

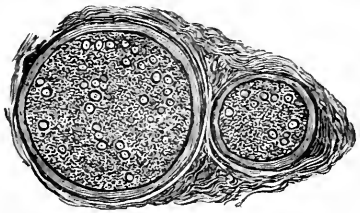


Fig. 136.—Plantar Nerve, Simple Atrophy in Tabes.

it, just as the parched and dying turf is the result of the fire which has swept over it. Neurology knows no tertiary syphilis, but the disease departs, leaving a trail behind it which sets in motion the processes of decay and death of the parts. This is what happens in locomotor ataxia, which is the Parthian shot of the conquered infec-

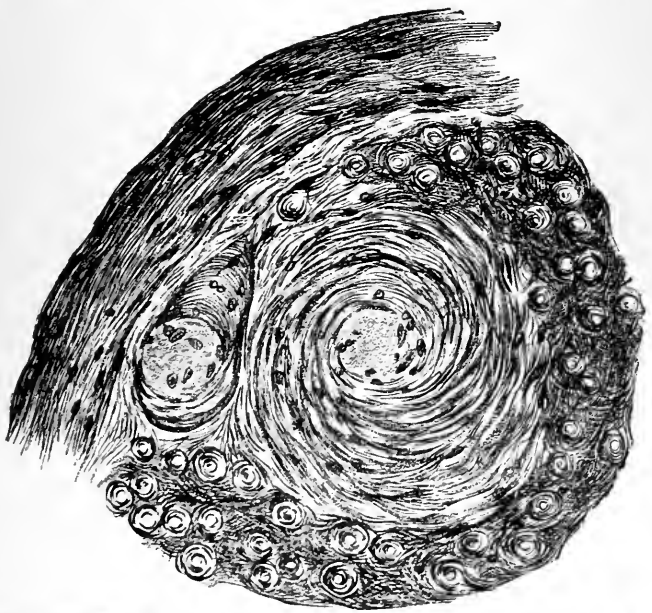


Fig. 137.—Ulnar Nerve, Third Stage Tabes, Atrophy with Proliferation of Connective Tissue (Oppenheim).

tion. Syphilis often invades the nerve centres and frankly shows itself in the form of inflammatory and gummatous exudates, but often it does not betray its presence and does its final work quietly through years of apparent health. All the time, however, its poison

is at work instituting a tendency to death and degeneration in certain parts of the nerve centres. The parts which are usually first selected are the posterior spinal ganglia, and, particularly, the neuraxons which pass from the nerve cells of these ganglia into the posterior roots and columns of the spinal cord. Locomotor ataxia, therefore, is not primarily a sclerosis of the posterior spinal columns, but of the peripheral sensory neurons. It is true that the posterior spinal ganglia are not always so seriously diseased as the posterior columns of the cord. This, however, is because the severity of the disease is first shown in the neuraxons and their collaterals, just as in alcoholic neuritis the peripheral parts of the motor neuraxons are most and earliest affected by alcohol. As the disease extends, it involves both the peripheral and central parts of these sensory neurons; that is to say, both the sensory fibres and the posterior spinal roots. Still later it attacks other portions of the nerve centres, so that in the last stages much of the nervous system is diseased. The reason why the peripheral sensory neurons are especially affected rather than other parts seems to me to be this: the syphilitic poison is brought to the nerve centres in the blood, whence it passes into the lymphatic sheaths of the blood-vessels or is thrown out upon the serous membranes in the subdural sacs. In the attempts of nature to get it out of the spinal canal and eliminate it, the poison is carried along the serous sheaths which surround the cerebro-spinal nerve roots. The nerves, as they pass out from the spinal cord, are covered with three membranes: the dura mater, the arachnoid, and the pia mater. The dura and the arachnoid surround them less tightly than the other. The dura becomes fused with the connective tissue supporting the nerves as they pass out, and the arachnoid becomes fused with the epineurium and perineurium. Now, fluid injected into either the subdural or subarachnoid spaces passes readily along the nerves for some distance (Macewen), and syphilitic exudate in the subarachnoid and subdural spaces of the spinal cord will thus have a tendency to infiltrate along these sheaths, but as it passes out of the vertebral canal or cranial cavity it meets mechanical obstacles, owing to the constriction of the parts, and there is therefore a certain damming up or accumulation of the poisonous material at these points of exit. Generally at this point it meets with the posterior spinal ganglia, a highly organized structure with special vascular supply, and it therefore naturally deposits its poison upon this part, which furnishes much more opportunity for mischief than the non-vascular anterior roots. It is a fact that many of the initial symptoms of locomotor ataxia are thus connected with such

troubles as would occur from an exudate trying to get out along the course of the spinal or cranial nerves. A frequent initial symptom, for example, is palsy of one of the third nerves or of the sixth or seventh nerves, due to exudates clinging around their roots. Still more frequently the initial symptom is a neuralgic pain in the course of the sciatic plexus, due to the effects of this poison upon the ganglia lying in the lumbar intervertebral spaces.

I assume in this description that it is a syphilitic poison which is always at work as a cause of tabes dorsalis. This, however, is not necessarily the case, for it may be the infection of other diseases, and the results of other poisons, such as ergot, pellagra, lead, etc., are to be explained in the same way. There is an inadequacy of the lymphatic and venous systems to thoroughly rid the spinal canal of the poisons that lie in it; the body of the cord is cleared, but the roots do not get rid of the poison. In a word, I would say that locomotor ataxia is a post-infective degeneration which first attacks the posterior spinal ganglia or corresponding cells of the special senses, due to a prolonged poisoning of these parts by the toxins of the infection, whatever this may be.

As to why this process attacks some and not others, it can only be said that certain people are born with defective power of resistance as regards their nerve centres, and that others induce this defective state by physical and other excesses.

The *diagnosis* is not difficult in the advanced stages. In the first stage the disease has to be distinguished from hereditary ataxia, multiple neuritis, chronic myelitis, spinal tumor, spinal syphilis, general paresis, and neurasthenia. In hereditary ataxia the age, the history of the disease, and the absence of lightning pains are usually sufficient to distinguish it. Multiple neuritis, in its sensory or pseudo-tabetic form, sometimes resembles closely locomotor ataxia. The differential points are given in the sections devoted to that disease. In myelitis there is more paralysis, generally exaggeration of reflexes, and an absence of disturbance of special senses.

The diagnostic criteria of locomotor ataxia in all cases are the presence of lightning pains, numbness of the feet, loss of knee jerk, ataxia of station and gait, without much loss of muscular power, the presence of the Argyll-Robertson pupil, the history of syphilis, and the slow onset of the disease. A *lost knee jerk*, *lightning pains*, and *stiff pupils* are usually quite enough to assure a diagnosis.

Prognosis.—In the first stage a small percentage may have the disease stopped and get practically well. After the second stage a cure is impossible, but great improvement may be secured and the patient made relatively comfortable for years.

In the third stage little can be done except to relieve the symptoms, but life may be prolonged. Death usually occurs from some intercurrent malady, or from kidney disease caused by the bladder trouble. Patients very rarely indeed die from the disease itself and its various "crises."

Treatment.—The treatment of locomotor ataxia is a subject the discussion of which cannot be made dogmatic, for the treatment depends very largely upon the patient and the stage and cause of the disease. My experience is that any treatment depends enormously upon one's opportunities of getting the patient in the earliest stages. Supposing this be done, the first thing is to be quite assured that there is no trace of a secondary (or exudative) syphilis underlying the trouble. If some of the symptoms are caused by such exudate, inunctions of mercury, warm baths, and iodide of potassium should be given vigorously and persistently.

I have seen no proof that inunctions are better than the internal use of mercury, but they are recommended by authorities eminent in neurology if not in cutaneous sensitiveness. I usually prescribe the bichloride in gr. $\frac{1}{20}$ doses and combine it with tincture of iron. The iodide of strontium is often a grateful change from potash. It is to be given in daily doses of about 60 grains, but this may be increased to 600 grains or 900 grains daily, with good effect. Since it is a fact that at times there are syphilitic exudates along with the true degenerative process in tabes dorsalis, this kind of treatment will occasionally give some good results. In the great majority of cases, however, mercury and potash do no good and they may actually do harm by hastening on the downward course of the disease; hence mercury in particular should be given with great watchfulness, and if improvement does not appear within six weeks it should be suspended. My own experience and the careful investigations of my friend, Dr. Collins, show that antisyphilitic treatment pursued at the time of infection may hasten the onset of the disease. It has been observed by others also that excessive mercurization tends to produce a neurasthenic state most prejudicial to the patient and one which may even lead to a certain amount of neuritis. When the physician has assured himself that any possibility of relief from mercurials or iodide is not to be hoped for, these drugs should be dropped; if they do benefit the patient, they should be repeated at intervals of three months. Along with these first medicinal measures, the physician should prescribe something which is much more important, and that is simply *rest*. Every patient with locomotor ataxia should at once have the importance of rest strongly impressed upon him, and the prescription of sixteen weeks

in bed is sometimes advisable. Equally good results can be usually obtained, however, by obliging the patient to go through a simple and regular life, involving no walking and no work. Institutional life for three months is of enormous advantage. It is a rule to which I have seen hardly an exception that tabetic patients brought to the hospital improve in a striking way simply from the quiet routine of life there, and despite the thinness of city milk and the odorous strength of hospital eggs.

The drugs which are at this time used to help in the cure are mainly the nitrate of silver, the tincture of iron, the preparations of phosphorus. I have not been able to convince myself that arsenic, strychnine, gold, ergot, barium, or aluminium do any good, although these drugs are all recommended by high authorities. The various preparations of the phosphates, such as glycerin phosphate of lime, the hypophosphites of lime, phosphoric acid, seem to me to be of some benefit. Strychnine occasionally does good in small doses, but in large doses it may lead to disastrous results and it should always be given with caution. A great many other drugs may be given for the relief of symptoms. For the pain, phenacetin is the drug which gives the most satisfaction excepting morphine. It may be combined with bicarbonate of sodium, with codeine, or with some of the other coal-tar products, such as antipyrin, antifebrin, etc. A teaspoonful of baking-soda internally will sometimes stop the pains. Hoffman's anodyne and cannabis indica may be tried. For the bladder trouble, the fluid extract of buchu in doses of twenty drops, combined with ten drops of the tincture of hyoscyamus, is very helpful. Small doses of strychnine may be combined with this. Small amounts of strychnine can always be given for the sexual weakness, but the dose should never be made a large one. For the gastric crises, nothing is so good as a hypodermic injection of morphine, and for the severe crises of pain an occasional hypodermic of morphine should be given. The locomotor ataxic, however, who becomes addicted to the use of morphine for his pains is indeed in a hopeless condition. In persistent constipation the diet should be light and mainly of liquids such as milk, malted milk, broths, etc. In persistent diarrhœal states I have found ichthyol of use. This drug also relieves the pains.

The annoying insomnia is to be treated by fresh air and seashore life. If drugs must be used, bromide of lithium with a few grains of chloral, and paraldehyde in not over thirty-drop doses, are the best.

In neuralgia of the rectum and bladder, suppositories containing iodoform and belladonna, or codeine, or antipyrin, may be used.

Sometimes simple gelatin or gluten suppositories act very well. Some of the cases of rectal neuralgia or hyperæsthesia are due to insufficient clearing of the lower bowel when a movement occurs, and if the patient washes out the bowel with a pint of warm water after each movement he is very much more comfortable.

There is no diet which has a specific effect upon locomotor ataxia, but the patient should be given those foods which are non-fermentative and digestible. Nitrogenous and fatty foods should be prominent.

Hydrotherapy is of considerable benefit. The most efficient of the single measures is the lukewarm bath at a temperature of about 95° F. for ten or twenty minutes daily. After the bath it is well to have a little cold water poured over the back and then the patient should be diligently rubbed. In most cases a simple lukewarm bath is quite as effective as anything. In others the patient feels better if there is added to it some slight stimulant to the skin—a tablespoonful of pine-needle extract, or a regular pine-needle bath may be given. The Charcot douche given in moderate strength is helpful in cases that are not advanced or particularly weak. I have some hesitation in recommending any special watering-places or cures. I have had patients return benefited from the Hot Springs of Virginia and other American resorts. In Europe, the baths at Lamalou, France, and at Nauheim, Germany, have some reputation. Hot baths are sometimes injurious, and bathing may be overdone by the ataxic.

Electricity is of use from its general tonic and reflex effects, and perhaps exercises some direct influence on the diseased process. Strong galvanic currents (15 to 30 ma.) should be applied along the spine, through the trunk, and down the legs and arms. The combined galvanic and faradic current is even better, given in the same way. The faradic brush should be applied over the extremities and along the back.

The actual cautery is efficient in stopping pains. It should be applied to the back as often as twice a month at least and sometimes twice a week. Dry cups may be applied rapidly and in great number (80 to 100) along the spine and along the course of the sciatic nerves. In very painful cases occasional wet cups and leeches are useful. Blisters and various forms of counter-irritant sometimes do good.

Suspension by the neck and arms is helpful in some cases. It is best adapted to persons in the second stage and to those who have a good deal of bladder trouble and pain. It is of little value in the paralytic stage, and must be used with care in the early stage and when patients are large and heavy. Suspensions should be given

for from one to three minutes three times a week until twenty-five or thirty are taken. After three months a second course may be given. The treatment of locomotor ataxia by systematic exercises, known as the Fraenkel method, is one that of late has been considerably used. It consists in having the patient go through regular exercises which teach him to co-ordinate the different groups of muscles of the trunk, legs, and arms. A list of the exercises such as I have used for some time is given in the Appendix. The Fraenkel method is one which can be used with advantage with persons who are passing into the second stage of tabes and in whom the disease is not making progress. It often enables the patient to walk better and use his arms better, but it does not especially affect the progress of the disease.

Finally, it has seemed to me that those sufferers from locomotor ataxia do best who persistently and courageously fight against their malady. Those who, despite suffering and discomfort, will three or four times a year take some form of treatment, medicinal, hydrotherapeutic, or electric, such as will have some beneficial effect upon their general nutrition, and such as will buoy up their hopes and improve their mental condition, are quite sure to be rewarded and after a hard fight emerge into a state of comparative relief from their symptoms and secure a measurable degree of rest from the progress of their disease.

SPASTIC SPINAL PARALYSIS (Lateral Spinal Sclerosis). Little's Disease.

This is (*a*) a term used to describe a form of paraplegia caused by chronic myelitis, and (*b*) a congenital disorder, known as Little's disease, in which there is sclerosis of the lateral columns of the cord.

The special interest attached to this form of disease, on account of the controversies concerning it, leads me to say a few words about its history. Between the years 1846 and 1877 an English surgeon, Little, published a number of articles on a disorder which he termed "congenital spastic rigidity of the limbs." In 1873 and again in 1879, Dr. E. C. Seguin, of New York, described a condition which he termed "tetanoid paraplegia." After the first article of Dr. Seguin, to whom priority belongs, Professor Erb and Professor Charcot independently published articles in the year 1875 upon what Erb called "spasmodic spinal paralysis" and Charcot "spasmodic dorsal tabes."

Starting from the writings of these three authorities, there developed in the course of a few years a description of the disease

which became known as "spastic spinal paralysis" or "lateral sclerosis." This for a long time was accepted as an independent malady by most writers. Of late years, however, its real existence has been strenuously denied, and the cases supposed to represent this disease were asserted to be either forms of dorsal myelitis or the result of some cerebral defect. Recently both French and German writers have revived the work of Little, and, having supplemented his observations with their own, have rehabilitated spastic paralysis into a separate disease again, giving it the name of "Little's disease." *

Etiology.—The new spastic spinal paralysis, or "Little's disease," is an affection which is always of congenital origin and is due, it is supposed, to a lack of development of the pyramidal tracts. This lack of development leads to a sclerosis of the lateral columns of the spinal cord and to symptoms of rigidity of the legs and arms, exaggeration of the reflexes, with some real muscular weakness and atrophy. The disease is always of prenatal or natal origin, being due to some developmental defect or, as Little supposed, to premature and forced deliveries. It may also be a hereditary family disease. Through these causes the pyramidal tracts cease to grow, or, at least, this process is greatly delayed.

Symptoms.—The malady appears within a short time after birth, usually within a year, but it may be delayed in family types to the fifth year or even later. Some cases of Little's disease may, it is believed, develop as late as after maturity. It is not my purpose to give a description in detail of the symptoms of this trouble, because they are given under the head of cerebral diplegia or birth palsy. The only difference between ordinary cerebral diplegia and the disease under present consideration is that in this latter form there are no marked mental defects; the child is not small headed and idiotic, nor does it have epilepsy or cranial nerve palsy or hydrocephalus. The brain seems to be spared except so far as its motor functions are concerned. It is convenient to separate this type of disease from the ordinary spastic cerebral palsies with mental defect, for the reason that the future of these cases is in some instances more hopeful. As they mature, the lateral columns occasionally gain in development and some increase in the strength and control of the limbs is obtained. I base this statement upon the experience of

* Sometimes a spastic paraplegia develops quickly; after a few weeks the symptoms improve and the patient gets well; this has been called "hypertonic paralysis." There is here, however, simply a slight grade of myelitis or meningo-myelitis, and no separate name is required to show what is the matter.

others. In several cases of Little's disease at the age of fifteen to twenty-five which I have seen, there has been no marked improvement. Mentally, however, these patients are often very bright.*

Children with this trouble on trying to walk are obliged to cross one leg in front of the other as they are helped along, giving them a characteristic "cross-legged" progression. The arms are less affected than the legs. The facial and throat muscles may be slightly involved. There is no pain. In some cases the disability increases as the child grows older, owing to the greater size and clumsiness of the patient. The arms become much stiffened and contractured, and the hands are flexed so that the patient can neither walk nor help himself. Epilepsy and mental deterioration also may develop at the time of puberty or adolescence.

Prognosis.—The mild cases that learn how to walk and can use the arms and hands may grow up, slowly improving, and reach a good age and a fair degree of health. The severer cases rarely reach adolescence, but grow gradually more helpless and generally succumb to some intercurrent disease before they are twenty.

Diagnosis.—The disease is distinguished from ordinary cerebral diplegia (birth palsy) due to brain lesion by the absence of epilepsy, mental defects, and microcephalus.

From compression myelitis, the involvement of the arms, and the absence of pain and disturbance of sphincters are distinctive. Hereditary spastic paraplegia runs in families, begins at the fourth or fifth year, and involves chiefly the legs.

Treatment.—This is altogether one of mechanics and attention to nutrition. The limbs must be persistently *masséd;* tenotomies should be performed so as to straighten the legs; constant voluntary effort to use the stiffened muscles should be made. Braces, roller crutches, etc., should be used. Patience is often greatly rewarded in this disease.

Hereditary Spastic Spinal Paralysis.—Spastic paralysis in very rare cases is found to run in families, affecting different members of many succeeding generations. In the cases described, it begins at about the age of five, affects only or mainly the legs, runs a very slow course, is not accompanied by pain, ataxia, or visceral symptoms; and runs a course lasting twenty or thirty years. Dr. Bayley, of Philadelphia, has described a family of typical cases.

* It is due to American neurology to say that Dr. Seguin as long ago as 1879 said: "It is possible that tetanoid paraplegia in young children may be due to deficient cerebral development and consequently agenesis of certain tracts of the cord."

THE COMBINED SCLEROSES.

By the combined scleroses is meant those forms of degenerative sclerosis in which both the posterior and lateral columns are involved. There are several diseases in which combined sclerosis exists. They are:

1. Combined scleroses of profoundly anæmic and toxic states (Putnam's type).
2. Hereditary spinal ataxia (Friedreich's ataxia and hereditary ataxic paraplegia).
3. Combined scleroses complicating general paresis.
4. Accidental forms (Gower's ataxic paraplegia).

There are many cases reported in literature of combined scleroses but the clinical pictures vary very greatly. These cases are probably in the most part forms of chronic myelitis or meningo-myelitis with ascending and descending degeneration. Marie has shown that the vascular supply of the spinal cord is such as rather to favor the development by extension of sclerosis in the lateral and posterior columns from a chronic leptomeningitis, and his suggestion that many of these cases are perhaps of syphilitic origin accords with my experience and conviction. Some years ago, Gowers described a disease that he called ataxic paraplegia, the lesion in which, he believed, lay in the lateral and posterior columns. Most of the cases which belong to this clinical description I think can be properly classed either with the cases of locomotor ataxia, of multiple sclerosis, or of some form of chronic myelitis. It seems to me, therefore, inadvisable to encumber our neurology with a description of this disease. There is, however, a hereditary form of ataxia with paraplegia which belongs to the group of congenital or family diseases and is closely related to Friedreich's ataxia; and there is a form of combined sclerosis in which some ataxic and some sensory and motor symptoms develop associated with pernicious anæmia and certain other cachectic states, but neither of these is the disease commonly spoken of as ataxic paraplegia of Gowers, which is, I repeat, no disease at all. Strümpell and many others have reported cases with autopsies showing combined scleroses, but there is no clinical picture that can yet be attached to such findings except those I have above indicated. Hence, of all the combined scleroses, it is only hereditary ataxia and the combined scleroses of anæmia that have practical clinical interest.

1. The Combined Scleroses of Pernicious Anæmia and Cachectic States (Putnam's Type).

This form of disease, not so rare as has been supposed, was first described by Dr. J. J. Putnam, later by myself; and in quite recent times has been expanded and placed in relation with pernicious anæmia by a number of observers.

Etiology.—In the original cases the patients were mostly women and the ages ranged from forty-five to sixty-four years. Two of my cases were men, one was a woman; all were over fifty years of age. A history of possible lead poisoning was obtained by Putnam. In my experience profound or prolonged malarial toxæmia was the only factor I could discover. The causes of pernicious anæmia must be placed in the list of the causes of this form of sclerosis. Disease of the suprarenal capsules and probably any very prolonged and profound toxæmic state may lead to the double degeneration of the cord. Not all cases of pernicious toxæmia or anæmia, however, cause these changes. Hence the element of a neuropathic state must be admitted.

Symptoms.—The symptoms begin generally with numbness of the extremities, followed by progressive enfeeblement, and ending in a paraplegia. Great emaciation and anæmia are present, and there is often an obstinate diarrhœa. No paralysis of any special groups of muscles occurs until the final paraplegia sets in. There are in some cases anæsthesia and ataxia, but spastic symptoms, with exaggerated knee jerk and ankle clonus, are the more common. Lancinating or girdle pains are very rare. The arms are affected, but less than the legs. The visceral centres are not affected till late. The vision and other special senses and speech are not disturbed. Mental symptoms approaching dementia occur in the terminal stages in some cases.

The general course is that of a rather rapidly progressive affection causing paræsthesia and sometimes anæsthesia of the extremities, especially the lower, with progressive weakness of the extremities. This is associated with very profound anæmia, general muscular emaciation, diarrhœa, ending in a paraplegia.

Pathological Anatomy.—The pathological appearances of the spinal cord as described by Putnam correspond to my own observations: in all the cases two sets of changes in the cord are recognizable: one of older date, consisting in a relatively dense sclerosis in the posterior columns and in the lateral columns (mainly confined to the pyramidal tracts); and one of subacute character, and evidently of quite recent occurrence. This subacute process is, as regards the white columns, partly in new tracts, partly around the borders of the more dense sclerosis, and is chiefly characterized by the

peculiar perforated appearance which indicates a somewhat rapid destruction of nerve tubes, with the œdematous distention or destruction of the intervening septa, associated with the formation of granule cells.

In the gray horns the degenerative change (partly recent, partly of older date) is indicated by a disintegration of nerve cells.

In the cases of distinctly pernicious anæmia the terminal softening is not described and there is only a well-marked sclerosis involving the posterior and lateral columns and more marked in the upper regions of the cord (Fig. 138).

The *prognosis* is not good, but there are more favorable cases than those first observed, and I have a patient who has kept fairly well for over six years. Two others died within two years.

The *diagnosis* is based upon the age of the patient, the presence of profound anæmia and perhaps of a malarial history, the paræs-

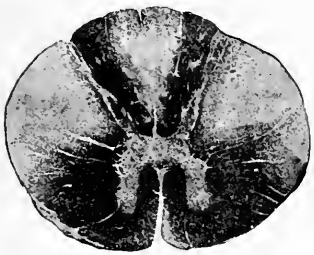


FIG. 138.—THE SPINAL CORD IN COMBINED SCLEROSIS FROM PERNICIOUS ANÆMIA, DORSAL REGION. The columns of Goll, crossed pyramidal tracts, and cerebellar tracts are affected.

thesia, slight ataxia, marked and progressive weakness and emaciation, tendency to obstinate diarrhœa, and finally the rather sudden paraplegia.

The *treatment* should consist of quinine, arsenic, iron, bone marrow, and possibly suprarenal-capsule extract. Besides this, the patient should have the most nourishing food and a stimulating air, free from malaria.

HEREDITARY SPINAL ATAXIA (FRIEDREICH'S ATAXIA).

Introduction.—There are three forms of ataxia of congenital and often family origin. They are: Hereditary spinal ataxia, or Friedreich's ataxia, hereditary cerebellar ataxia, and hereditary ataxic paraplegia. They are quite similar in cause and mode of development. The difference in symptoms depends upon the fact that the defect develops in the one case mainly in the posterior and lateral columns of the cord, in the second in the cerebellum, and in the third mainly in the lateral columns.

Friedreich's ataxia, the most common of all the forms, is a chronic degenerative disease mainly affecting the posterior and lateral columns of the cord.

Clinically the disease is characterized by ataxia beginning in the lower limbs and gradually involving the upper limbs and the organs of speech. Curvature of the spine, talipes, vertigo, and finally paralysis and contractures appear. The knee jerk is, as a rule, absent. There is but little pain or anæsthesia, and optic atrophy and visceral troubles are usually absent.

Etiology.—The fundamental factor in predisposition is an inherited or connate lack of development of the spinal cord, more particularly of the posterior columns and pyramidal tracts. This condition is inherited directly sometimes, but indirectly as a rule; that is to say, the parents or other members of the family usually show simply a neurotic history, and it is in only a minority of cases that there is a history of ataxia in the direct line of ancestry.

The more frequent condition is this: the parents or grandparents have some neuroses, such as insanity, inebriety, or great nervous irritability; then the ataxia occur in the children of the next generation. Sometimes in a single family the uncles and nephews or cousins may be found to have the disease. Hence the name "family ataxia," used by some writers. There are a good many cases in which the parents were apparently perfectly sound and healthy. Yet it is most probable that the sufferers from Friedreich's disease inherit a tendency to degenerative processes from some of their ancestors. This degenerative tendency may have been shown in those ancestors in a very slight degree. The patients rarely have locomotor ataxia, though this has been observed in a few cases. The children of locomotor ataxics do not have Friedreich's ataxia except in the very rarest instances.

Syphilis in the parents is an element in some, probably in most cases. Habitual intemperance in parents undoubtedly is a factor sometimes; much more rarely consanguinity and tuberculosis act as predisposing causes of degeneration.

More cases have been observed in America than in any other country; while the fewest have been reported from France. The disease develops at about the time of puberty, most cases occurring between the ages of six and fifteen years. It is not very rare, however, for symptoms to develop even in infancy, though some of the cases reported at this time were probably of a syphilitic character. In a given family the disease, as a rule, strikes the older members first, but the younger members are attacked at a relatively earlier age. The most typical time of development is a rather late one, *i.e.*,

after twelve years of age. The disease may come on after maturity. In American cases the age of development of the disease has been rather earlier than the average. The male sex slightly predominates, its proportion being about sixty per cent. In America the female sex has, however, been more affected (3 to 2). The patients are the children of the laboring and agricultural classes. They have been found in the country oftener than in crowded cities. The families have often been large, but this is not always the case, especially in American cases. Nursing at the mother's breast is thought to have been an exciting cause. Usually the disease appears after infectious fevers, such as diphtheria, variola, and typhoid.

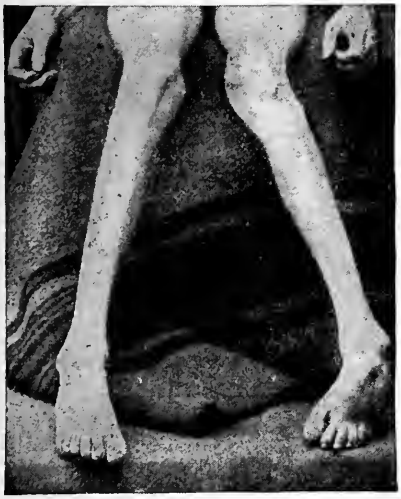


FIG. 139. — FRIEDREICH'S ATAXIA, showing deformities of legs in the late stage.

Symptoms. — The patient first notices an uncertainty in the gait and some feebleness in the lower limbs. These symptoms gradually increase until they interfere seriously with progression, and force him to leave off active work. With this there may be some slight pains or numbness in the lower limbs, and an examination will show, within a year or earlier, that the knee jerk is gone. After five or six years the arms become affected with inco-ordination, and a little later bulbar symptoms, such as thick or scanning speech, and often nystagmus, appear. During this time the patient suffers little pain and has no trouble with the bladder or rectum. Vertigo and headache are often present. Dorsal flexion of the toes, talipes varus or some other form of clubfoot, and lateral curvature of the spine are often observed (Fig. 139). Oscillation of the head and choreiform or inco-ordinate movements of the extremities may develop. As the disease progresses the legs become weaker, and finally paraplegia, with contractures and muscular wasting, sets in. The disease makes slow progress; often it remains almost at a standstill for years, and the patients usually die of some intercurrent disease, such as phthisis or an infectious fever.

Among the rarely observed symptoms are tremor, spasms, decreased electrical irritability, muscular atrophy, vasomotor paresis,

polyuria, glycosuria, anæsthesia, fibrillary tremor, choking attacks, ptyalism, strabismus, diplopia, blepharospasm, a slight degree of

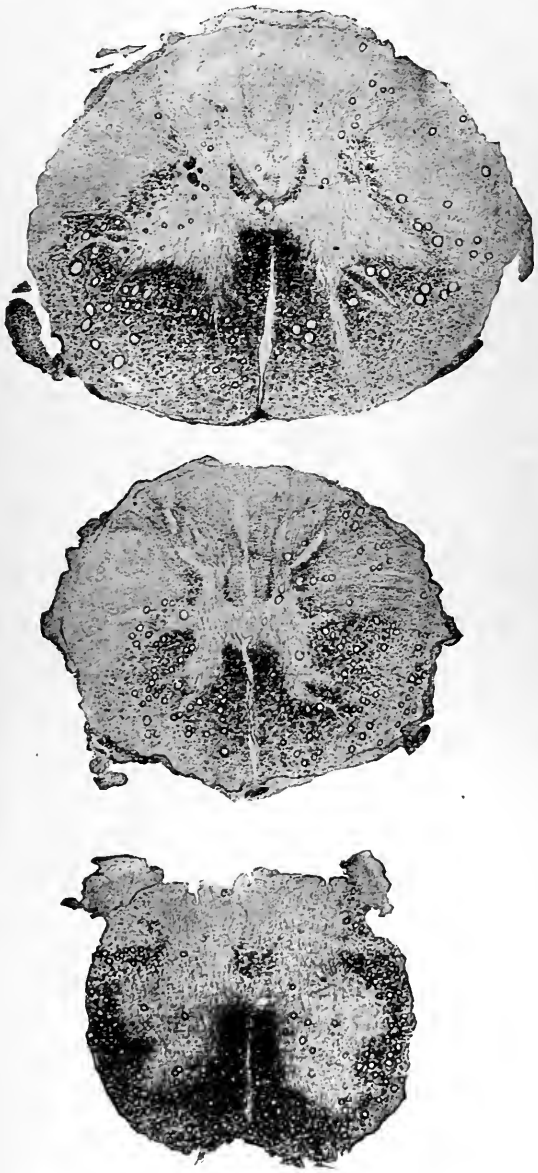


FIG. 140.—SPINAL CORD IN A LONG-STANDING CASE OF HEREDITARY ATAXIA, CERVICAL, DORSAL, AND SACRAL REGIONS. Besides the sclerosis, the cord is filled with small vacuoles which are dilated perivascular spaces.

ptosis, sluggish pupils, tachycardia, profuse sweats, impotence, slight vesical incontinence, fragilitas ossium. Many of these symptoms are, however, exceptional and accidental.

The major and essential symptoms are: (1) ataxia, beginning in the lower limbs and extending to the arms and tongue; (2) peculiar rolling, ataxic gait, ataxia gradully involving the arms; (3) disturbances of speech; (4) talipes and spinal curvatures; (5) gradual development of paraplegia; (6) loss of knee jerk; (7) absence of cutaneous anæsthesia, of bladder troubles, of eye troubles except nystagmus, and of severe pains; (8) the development of the foregoing at about the time of puberty.

Pathology.—The lesions of importance are found in the spinal cord and medulla only. The cord is usually small, flattened, and apparently congenitally imperfect in development. In some cases two central canals have been seen. A sclerosis exists throughout the whole length of the posterior and lateral columns, sometimes extending to the anterior columns (Fig. 140). The sclerosis is most marked in the postero-median columns, which are always affected *in toto*. The postero-external column is less involved and there is often a narrow strip of healthy tissue between the posterior horn and the sclerosed area, also between the posterior gray commissure and the diseased parts. The posterior-column sclerosis is usually most marked in the lumbar region. In the lateral columns the sclerosis always affects the crossed pyramidal tracts. The direct cerebellar tracts and the so-called ascending antero-lateral tract are diseased in some cases, but apparently not in all. In a few instances the anterior median columns are involved. A zone of healthy tissue is often found between the sclerosed pyramidal tracts and the posterior horn. There are no important changes in the gray matter. Some chronic leptomeningitis, especially on the posterior surface, has been noted. The medulla shows some traces of extension of the sclerosis, but the involvement of the cells of the hypoglossal nucleus is probably the most significant change. The brain exhibits no changes of importance in relation to the symptomatology of the disease. The posterior nerve roots are extensively sclerosed, the anterior roots less so, and the peripheral nerves show some degenerative changes. The peripheral nerves are much less involved than in tabes dorsalis. It is asserted that the sclerosis in the cord is really a neuroglia proliferation—a gliosis—and there is no doubt a large amount of neuroglia proliferation in the diseased areas. Curious vacuoles were found in one case examined by myself. They were due to dilated perivascular spaces (Fig. 140).

Course and Prognosis.—The disease is a progressive one, though

it may be stationary for a long time and may even show temporary improvement. The longest period of duration of the disease on record is forty-six years and the shortest two years, the average being fifteen or twenty years. Death occurs from some intercurrent disorder.

Treatment.—A quiet life, good food, and favorable hygienic surroundings are the main therapeutic helps. Arsenic and various nerve tonics may be of temporary benefit. My cases and some of the French cases were benefited by suspension by the neck in a Sayre apparatus. If the disease appears in one member of a family, effort should be made to prevent its appearance in others. The infant should not be nursed by its mother; special care should be taken to prevent its getting any infectious fevers and to prevent it from receiving any falls or blows. Its life should be exceptionally quiet, so far as physical exertion goes.

3. Hereditary Ataxic Paraplegia.

This is a hereditary disease and sometimes a family one. In six cases observed by myself, there were no other instances in the families. All my patients were young women. The disease began between the ages of twelve and sixteen, but sometimes the patients could remember that they had never been so quick of foot as other girls. A neurotic heredity existed, but no syphilis or alcoholism. The exciting cause is not known.

The disease begins with a stiffness and weakness of the legs. Associated with this is a decided ataxia. This is occasionally more cerebellar than spinal, but in most cases it is shown by awkwardness in posing and in moving the limbs in different definite directions, and in walking a line. There are decided exaggeration of reflexes and ankle clonus, but no painful spasms, no pains, and no distinct anæsthesia; there is some paræsthesia. The trouble is much the most marked in the legs, but the arms are slightly involved, the face and cranial nerves not at all. In one case optic neuritis was present.

The sphincters are not involved, the general nutrition is good, the mind is clear. The disease in most cases progresses very slowly. One of my patients is married and continues in fair health.

In some cases, evidences of degeneration and breaking up of nerve centres occurs, just as in the terminal state of Friedreich's ataxia. However, I have one patient who has had the disease in a mild form for twenty years and is now quite comfortable.

Hereditary ataxic paraplegia is more allied to the spastic para-

plegias than to Friedreich's disease, and it has, like that malady, a rather more favorable course—so far as my limited experience shows. I know of no autopsies made on these cases, but the symptoms point to a defect of the lateral columns and of either the cerebellum or the posterior columns.

4. Hereditary Cerebellar Ataxia.

Though not belonging in the group of spinal diseases, hereditary cerebellar ataxia is so closely related to those hereditary forms I have just been describing that I deem it best to give an account of it here.

This is a chronic disease beginning in early life, usually of hereditary or congenital origin, and characterized by an ataxia of cerebellar type associated with symptoms indicating the involvement of some of the cranial nerves. In course and symptoms it resembles to some extent hereditary spinal ataxia and belongs to the same class of diseases. The names of Fraser, Nonne, and Marie are connected with the first descriptions of this disorder and its differentiation from hereditary spinal ataxia.

Etiology.—The malady begins in early life between the ages of ten and thirty, developing, therefore, somewhat later than Friedreich's ataxia. The disease occurs rather more often in males than in females, but, like other family diseases, is usually transmitted by the female. Syphilis and a neuropathic constitution in the female have been noted in a number of cases.

Symptoms.—The first symptom is shown in a disturbance of the gait. This is indicated by clumsiness and stumbling and by a tendency to rolling and pitching like a drunken man. The patient tends to walk with the feet wide apart. He does not, however, show the "Romberg symptom;" that is, he can stand fairly well with the feet together and the eyes shut. There are inco-ordination and jerkiness in the movements of the arms and sometimes choreic movements. He sometimes also has oscillation and jerky movements of the head. When lying down the inco-ordination is very much lessened. The speech is hesitating, ataxic, and explosive. The eyes show jerky movements somewhat like those in nystagmus. An important symptom in many cases is the development of optic neuritis followed by optic atrophy and blindness. The knee jerks are usually exaggerated. The patient has no anæsthesia and suffers little pain, although he may have some headache. There is no disturbance of the sphincters. Mentally the patients are usually somewhat deficient, becoming either simply childish or actually demented. The

malady progresses, although not always steadily, the disease sometimes remaining stationary for some years. Eventually, however, the patient becomes bedridden and dies of exhaustion.

Pathological Anatomy.—The few autopsies which have been made show an atrophy of the cerebellum. In some cases this is macroscopic, the cerebellum being reduced to one-half or one-third its size. In other cases there is no naked-eye change, but there is found microscopically an atrophy of the cells of Purkinje.

Diagnosis.—The disease has not yet been sufficiently studied to enable one always to recognize it positively. Indeed, it is not unlikely that there are mixed or transitional forms of hereditary cerebellar and hereditary spinal ataxia. Furthermore, it is probable that in some cases small hemorrhages or nodules of sclerosis may occur after fevers during childhood, which may lead to the development of symptoms resembling the hereditary malady. However, the points of diagnosis, as far as established, consist in the hereditary history or family history of this malady. Next is the fact that it develops gradually, beginning with the ataxia characteristic of the cerebellar disease, but involving the arms as well as the legs. Again, there is always an increase of knee jerks, whereas in spinal ataxia these are lost. Then we find in this malady no pain or anæsthesia in the legs, and no involvement of the sphincters. The occurrence of optic atrophy and of mental defect help much to differentiate the disease from other types.

Treatment.—The disease is a progressive one and little can be done but to attend to the general nutrition of the patient. In one case, however, seen by me with Dr. Stillwell, it was found that the continuous use of antipyrin was accompanied by an amelioration of symptoms and seemed really to help the patient a great deal.

CHAPTER XV.

THE PROGRESSIVE MUSCULAR ATROPHIES AND MUSCULAR DYSTROPHIES.

The result of modern studies is to show that the anterior cornual cells of the spinal cord, the motor nerves and their terminal end organs, the muscles, form a trophic unit, and that the same degenerative disease may attack either end or any part of this physiological mechanism. There is a clinical and pathological unity in all the different spinal and muscular types of atrophies. But there are sufficient differences, also, to oblige us for convenience' sake to make certain classifications. Thus those disorders which attack chiefly and first the anterior horn cells and the pyramidal tract are called *progressive muscular atrophies;* those disorders attacking first the muscle tissue and its nerves are called *progressive muscular dystrophies* (Fig. 141). The progressive muscular atrophies of central origin may attack the motor-nerve cells of the eye, of the throat and lips, of the upper or lower spinal cord. In accordance with the level affected the disease has received different names. Sometimes the pyramidal tracts of the spinal cord are first and most involved. This has furnished excuse for another type. Then, again, while most cases of muscular atrophy are acquired, there is one type of it which is a hereditary one. Thus we find the disease classified as follows:

Progressive muscular atrophies of spinal or nervous origin.	Progressive ophthalmoplegia.
	Progressive bulbar palsy.
	Progressive muscular atrophy.
	Progressive hereditary muscular atrophy (leg type).
	Amyotrophic lateral sclerosis.

The progressive muscular dystrophies have also been much subdivided, but they are essentially the same disease, as will be seen later.

Of the muscular atrophies, I have already described ophthalmoplegia.

Progressive Muscular Atrophy (Progressive Spinal Amyotrophy; Duchenne-Aran's Disease).

This is a disease characterized by a slow, progressive atrophy of the muscles of the extremities and trunk, with consequent paralysis, not accompanied by any notable sensory disturbance, and due to a progressive atrophy of the motor and trophic cells in the spinal cord.

Etiology.—The disease affects persons in the middle period of life (twenty-five to forty-five). The extremes are fourteen and seventy years (Gowers). It is more frequent in males. Heredity is rarely, if ever, a factor. Great mental strain, exposure, traumatism, excessive use of certain groups of muscles, acute infectious diseases—especially typhoid, measles, cholera; childbirth, acute

Fig. 141.—Showing: 1, Segment of spinal cord with anterior-horn cell, end brush, and lateral tracts, the parts affected in progressive muscular atrophies; and 2, the muscle and its nerves, the parts affected in progressive muscular dystrophies.

rheumatism, syphilis, and, more than anything else, lead poisoning are causes. It may complicate locomotor ataxia. The causes, as may be seen, are much the same as those of bulbar paralysis.*

Symptoms.—The patient suffers at first from slight rheumatoid

* Among 28 of my cases 22 were males. The disease began between between thirty-one and forty in 10 cases; between twenty-one and thirty in 7; in 5 it developed before the age of twenty; and in 2 after the age of fifty, but in more after sixty. It begins rather earlier in women. In the majority, 20, the malady began in hand or shoulder and ran the classical course; in 3 it began in the legs and ascended; in 3 it began in the medulla; and in 1 in oculo-motor nuclei, speedily extending to the arms and legs (polio-encephalo-myelitic type). In 2, beginning in the medulla, it assumed the amyotrophic lateral sclerosis type.

The occupation of the patients is very suggestive. In 10 there was a history of excessive work with the arms, the patients being smiths, iron workers, bricklayers, boilermakers, horseshoers, stonemasons, tailors, barbers, locksmiths. In 3 there was a history of syphilis, in 1 of lead, in 2 of grippe and the puerperium, in 1 of infantile spinal paralysis.

pains in the shoulder or arm, associated with some feelings of numbness and weariness. Muscular wasting then begins to appear, and usually in one hand. The adductor longus pollicis is very early affected, also the thenar muscles and the interossei. The atrophy spreads from muscle to muscle, and does not follow the distribution of nerves, although the ulnar-nerve supply is most seriously disordered. The ball of the thumb becomes flattened, and the patient cannot abduct or flex it well. When the radial interossei are reached the forefinger cannot be abducted, and this is often an early sign. The disease gradually extends upward, attacking the flexors and extensors of the forearm, then the upper arm and shoulder. Meanwhile the hand has become thin and flattened, flexion of wrist and extension of fingers are lost, and a characteristic "griffin-claw" appearance results. After a time (three to nine months) the other arm begins to be affected. Occasionally there is a temporary remission.

FIG. 142.—SHOWING WASTING OF HANDS IN MUSCULAR ATROPHY.

In a few cases the atrophy begins first in the shoulders and arms, attacking the deltoid, biceps, and triceps, then extending downward to the hands. This constitutes the "upper-arm type."

If, as is usually the case, the disease continues to progress, it passes from the shoulder girdle to the deep muscles of the back, then downward, involving successively the hip and thigh muscles, the glutei, the crural extensors and abductors being oftenest chosen. The leg muscles may be finally involved, but they usually escape. The disease as it descends continues its progress in the trunk, involving the intercostals. It slowly ascends the neck also, and finally leads to paralysis of the diaphragm, or a bulbar palsy may set in.

It will be seen that the ordinary course of the disease is from the lower-arm muscle groups (ulnar and median) up to the shoulder group (middle cervical nerves), then down through the dorsal and lumbar nerves, rarely reaching the sacral groups. In very rare cases it begins in the legs and ascends.

Along with the wasting there are a corresponding weakness and

paralysis, but the paralysis is the result of the atrophy and does not precede it. Fibrillary twitchings of the muscles occur; the idiopathic muscular contraction caused by striking it a blow is very marked; myoid tumors are easily brought out. In some cases the muscles are flaccid and toneless, and the deep reflexes, knee jerk, and arm jerk disappear early (atonic atrophy), but in other cases the rigidity and tonicity of the muscles are increased, the knee jerks exaggerated, and we have tonic atrophy. This condition may be so marked as to make it resemble a special clinical type of progressive atrophy known as *amyotrophic lateral sclerosis.*

The electrical irritability of the muscles lessens to both galvanic and faradic currents, but no marked qualitative changes occur at first. Eventually we may get partial degeneration reactions, but these occur late in the disease, unless this runs a very rapid course, when fairly typical degeneration reactions may be got. A peculiar contraction of the upper limbs is sometimes produced by placing the negative pole of the galvanic battery over the fifth cervical vertebra, and the positive in the triangle just below the lower jaw (diplegic contraction of Remak). A peculiar palmar spasm is described by Voeter, caused by suddenly interrupting a faradic or galvanic current passed along the affected arm.

In typical cases of progressive muscular atrophy there is no anæsthesia; and when such symptoms develop the presence of peripheral disease or of syringo-myelia or spinal tumor must be suspected. The patients may suffer from rheumatic-like pains and from paræsthesias.

The affected parts often show excessive sweating and congestion and evidence of vasomotor disturbance. This may involve the face on one or both sides; one pupil may be larger than the other, due to irritation of the cilio-spinal centre. The iris reflex, however, is preserved, and the optic nerve is never involved.

The sexual power is often weakened, but the sphincters are not attacked. The urine shows variations in the amount of urea. There is usually an increase of lime salts.

Complications.—The most common complication is an extension of the process to the medulla, causing disturbance of speech and swallowing. Muscular atrophy complicates locomotor ataxia, but is rarely complicated by it. A high degree of spasm and rigidity of the legs, particularly, may occur, causing the condition known as amoytrophic lateral sclerosis.

Course and Duration.—The disease usually progresses steadily until it has reached an advanced stage, when it may stop. Remissions may occur earlier, however, and even some improvement take

place; the disease then ordinarily progresses again. It lasts from two years to thirty or more, but on the average not over ten or twelve years. Death usually occurs from pulmonary disease, owing to the weakness of the respiratory muscles. Sometimes the extension to the medulla and involvement of the muscles of deglutition and of the larynx are the cause of death.

Pathology.—The primary anatomical change is a degenerative atrophy of the cells of the central parts and anterior horns of the gray matter of the spinal cord. The atrophy gradually extends and involves the whole anterior horn. It also extends vertically, first down, then up. Along with this atrophy are degenerative changes in the lateral columns; consecutive to this there is atrophy of the anterior roots, peripheral nerves, and the muscles. The disease begins in the deeper parts of the anterior cornua, involving the central and median groups of cells. These are more concerned in nutrition and in the finer muscular movements of the extremities. Hence atrophy always precedes, or at least keeps pace with paralysis. The levels affected are the lower cervical and upper dorsal; but if the disease is extensive the dorsal, lumbar, and sacral cord are also involved. The affected part is nearly free from nerve cells, and those present are atrophied, their processes are short or absent, and the cell has lost its angular appearance. Sclerotic and pigmen tary changes are observed. The neuroglia and connective-tissue cells are increased in number, but there are no marked changes in the blood-vessels, though these may be much dilated. There is always some degeneration of the lateral columns, and this may be very complete. It is confined chiefly to the pyramidal tracts, but extends somewhat anteriorly into the mixed lateral column. It does not affect the cerebellar or ascending lateral tracts. The degeneration has been traced up into the brain as far as the internal capsule and even to the cortex. The anterior columns may be slightly affected. The posterior horns, columns, and roots are normal.

The affected muscles show various degrees of degeneration. They are pale and streaked with yellow, due to fatty deposits. Some fibres may be simply narrow and shrunken; others have lost their striation and become granular from deposit of fat globules or degenerated muscle elements; other fibres have lost their striations and appear as if filled with a homogeneous, glassy-looking substance containing a few fat granules (vitreous degeneration); others show a longitudinal striation. The interstitial connective tissue is increased and in places has taken the place entirely of the muscles. The capillaries and small vessels are distended. Healthy fibres

may be seen among the diseased. Changes have been found in the sympathetic nervous system, but they are unimportant.

The *diagnosis* has to be made from the progressive muscular dystrophies, chronic poliomyelitis anterior, syringomyelia, neuritis, and neuritic family atrophy.

In the muscular dystrophies there is commonly a history of heredity; the disease begins usually in childhood or adolescence. It attacks the lower limbs oftener; it is slower in progress; there are no fibrillary contractions, and the degeneration reaction does not occur.

Chronic poliomyelitis anterior begins suddenly and, having reached its height, does not progress, but remains stationary or improves. The paralysis occurs first, the wasting follows. It affects groups of muscles physiologically related, while progressive muscular atrophy attacks muscles only anatomically related. There are cases, however, which seem to be on the border line between the two diseases.

Syringomyelia is distinguished by the presence of peculiar sensory and trophic disorders.

Neuritis caused by lead poisoning is detected by the history of the case, its tendency to affect the extensors of the arm chiefly, and the absence of a progressive tendency. Sometimes, however, lead poisoning and palsy end in true progessive muscular atrophy.

Ordinary multiple neuritis is distinguished easily by its rapid onset and the presence of painful symptoms.

The hereditary or "leg type" of progressive muscular atrophy is characterized by its beginning in the legs, by a good deal of sensory disturbance, typical degeneration reactions, and hereditary or family history.

Treatment.—The patient should be well fed and have rest, quiet, and fresh air. Careful local faradization and galvanization of the spine and neck are indicated. Massage does no good. Hypodermic injections of strychnine in the affected member, gr. $\frac{1}{80}$ to $\frac{1}{30}$ daily, the internal use of arsenic, phosphorus, iron, quinine, and cod-liver oil sometimes are beneficial.

In a few cases with a syphilitic history, mercury and iodide of potassium have proved useful. The essentials of treatment are rest, electricity, strychnine locally, the administration of powerful tonics, and overfeeding. Nitroglycerin, morphine, atropine, nitrate of silver, chloride of gold and of barium, and the nitrate of uranium may be tried.

Progressive Hereditary Muscular Atrophy of Leg Type (Charcot-Marie Type).

This is a hereditary or family muscular atrophy of central (or neuritic?) origin, beginning in the legs and extending upward. It affects males more than females, but the difference is not great. It almost always begins before the age of twenty. It attacks first the muscles of the leg, not the foot, involving the peronei, then the extensors of the toes, then the calf muscles. The thighs escape till later. After some years the upper extremities and small hand muscles are reached. The shoulder and arm, neck and trunk muscles escape. There are occasionally fibrillary contractions; and always partial or complete degenerative electrical reactions. The patients complain of some pain and numbness, but there is no anæsthesia.

The disease runs a long course, with remissions, and resembles in prognosis the dystrophies. The outlook is better than in the arm type, but the disease is not curable.

Some authorities assert that the disease is due to a progressive degenerative neuritis. In the writer's opinion the anterior horns of the spinal cord are primarily attacked,* a view recently confirmed by Marinesco.

The *treatment* is the same as for the other forms of hereditary muscular atrophy.

Glosso-Labio-Laryngeal Paralysis (Progressive Bulbar Paralysis).

This is a disease characterized by progressive wasting and paralysis of the muscles of the tongue, lips, palate, and throat, due to an atrophy of the nuclei of the nerves supplying those parts.

Etiology.—It is a disease of the degenerative period of life, most cases occurring after forty and between that time and seventy. The disease begins later in life than spinal atrophy. It occurs rather oftener in men than women.† A neurotic heredity is sometimes noted. Exposure to cold and excessive use of the muscles in talking, mental strain, debilitating influences, lead, and syphilis are causal factors.

* The writer has seen the disease in a typical form in one member of the first generation, in two members of the second. A child of one of the latter had, at the age of two years, a typical attack of anterior poliomyelitis.

† While this is the usual statement, in my experience women suffer much oftener than men.

Symptoms.—The tongue is the part first affected. The patient speaks indistinctly and cannot articulate the lingual consonants *l*, *r*, *n*, and *t*. The tongue cannot be elevated and is protruded only a little distance. It looks scarred and wrinkled. The lips become weak and the patient cannot whistle nor make the consonants *p*, *b*, *m*, or the vowel *o*. The saliva begins to dribble from the mouth. Disturbance in swallowing soon develops. Hard solids are taken with difficulty, next fluids, while semisolids are generally managed best. The lips finally become so paralyzed that the mouth cannot be shut, and the lower part of the face is motionless and expressionless. The upper face wears an expression of anxiety and suffering, the saliva dribbles constantly, and the whole physiognomy of the patient becomes characteristic and pitiful in the extreme. The facial nerve may get somewhat involved. Articulation becomes almost entirely lost; the voice has a nasal twang from paralysis of the palate.

The patient has tired and uncomfortable sensations of dryness and stiffness about the throat. There is no pain or anæsthesia, but occasionally there is impairment of the sense of taste. The throat reflex is usually lost, so that tickling it causes no reaction.

Electric irritability is at first unchanged, but in the later stages partial degeneration reaction occurs. In rare cases there is a rapid pulse and still more rarely glycosuria.

The laryngeal reflex becomes weak, the adductors also, but abductor paralysis is rare.

The mind is not affected, but there are often an emotional weakness and tendency to tears—not entirely unreasonable in view of the distressing nature of the malady.

The disease is often the terminal stage of spinal muscular atrophy; it may be associated with the latter, with amyotrophic lateral sclerosis, or with ophthalmoplegia. All these types may occur together.

It runs a progressive course, with remissions of a few weeks or months. It lasts from one to three or four years. In one case it has lasted seven years.

The termination is eventually fatal. Death occurs through interference with swallowing, and inanition or a broncho-pneumonia or bronchitis may develop which ends the patient's life.

Pathology.—The primary lesion is found in the nuclei of origin of the hypoglossal, glosso-pharyngeal, vagus, and spinal accessory nerves. The raphe fibres and the anterior pyramids are also usually somewhat involved. There is sometimes atrophy of the cells of the facial nerve and of the nucleus ambiguus, which is the motor nucleus

of the vagus. The brunt of the disease falls, therefore, upon those more superficial or posterior nuclei which are representative of a continuation of the anterior cornual cells. If the disease is complicated with amyotrophic lateral sclerosis, or progressive muscular atrophy, or ophthalmoplegia, we find atrophy in the cord or ocular nuclei. The atrophic process is similar to that observed in the spinal disease.

The muscles of the tongue, and to a less extent the orbicularis oris and the throat muscles, show evidences of degeneration and atrophy. In some cases the tongue is not shrivelled, owing to the presence of a fatty deposit, and on account of this the disease has been divided into atrophic and paralytic types, but this distinction is unnecessary.

Diagnosis.—The disease must be distinguished from polio-encephalitis inferior, bulbar apoplexy, tumors, and softening, from multiple sclerosis, and from chronic lesions of the cerebral hemispheres causing pseudobulbar paralysis. It must also be distinguished from asthenic bulbar palsy. The slow onset, the progressive course, the bilateral character, the absence of involvement of sensory nerves, and the degenerative reactions are sufficient for a diagnosis. In asthenic bulbar palsy there is great paralysis, but none of the typical atrophy of the parts. It is important always to note whether there are ophthalmoplegia and spinal muscular atrophy associated with the disease.

Treatment.—The patient should be kept quiet; he must be overfed and given massage and electricity in moderation. The same drug treatment as in the spinal disease is indicated. Small doses of morphine, gr. $\frac{1}{24}$ to $\frac{1}{36}$, and of atropine may be given also. Electricity should be tried for a short time twice or even thrice daily, if possible. The faradic current may be used, alternating or combined with the galvanic. Galvanization of the neck and medulla appears to do no good. After a time it may be necessary to feed with a tube or even to do tracheotomy.

Asthenic Bulbar Paralysis and Asthenic Bulbo-Spinal Paralysis.

These names are given to a chronic and progressive disorder characterized by the symptoms of progressive bulbar paralysis or by the symptoms of this disease and of progressive muscular atrophy, the distinguishing features being that there is no muscular atrophy, that the cases often continue on for many years instead of going on progressively to a fatal issue, and also by the fact that on autopsy no easily distinguishable microscopical changes are found.

Etiology.—Little is known as to the cause of the disease. The majority of cases have been under the age of thirty, but a patient of my own was over fifty years of age and another over forty. It is sometimes associated with profound anæmia. Those causes which are found in progressive bulbar and spinal paralysis, viz., overwork, mental strain, are sometimes found here.

Symptoms.—The disease usually begins gradually and oftenest affects the muscles of the throat and face and of the eyes. A frequent symptom is ptosis of either one or both eyes. This may be followed by weakness or paresis of the muscles of mastication, defect in articulation, and difficulty in swallowing. The voice becomes nasal and the appearance of the patient very much resembles that of a case of glosso-labio-laryngeal paralysis. At the same time, with some ophthalmoplegia, there develop feelings of great exhaustion and extreme weakness in the arms and legs. The patient becomes incapable of anything but the slightest exertion and at times he is unable either to raise the arms or to stand upon the feet. The symptoms are characterized by remissions; after a patient has reached a point at which he is almost moribund, he begins to get stronger again and may slowly get into a state of comparative strength; then the symptoms slowly return. In this way the disease may continue for a number of years. The patient usually dies of exhaustion, but he sometimes recovers.

Pathological Anatomy.—In the half-dozen careful autopsies so far made, no lesion of the nervous system has been found, except microscopical changes in the cells of the motor nuclei.

Diagnosis.—The clinical characteristic which distinguishes this disease from progressive muscular atrophy and true bulbar palsy is the fact that there is no true atrophy of the muscles of the face or tongue or extremities, there are no fibrillary twitchings, and the course is irregular with remissions. Like these other diseases, however, asthenic paralysis is not accompanied by any disturbance of sensibility or any impairment of the sphincters. The patient may die in six months, or he may live for six years, or even recover.

The *treatment* consists in complete rest, careful attention to feeding, and the use of iron and arsenic, and, possibly, of quinine. Strychnine and muscular stimulants should be given with great care. Faradism is not advisable, but the use of a stable galvanic current is reported to have done good.

Amyotrophic Lateral Sclerosis.

(Spastic Form of Progressive Muscular Atrophy.)

This disease is one which has the closest possible kinship to progressive muscular atrophy which we have just described. There has been an enormous amount of discussion as to whether a distinct place should be given to the disorder. The question is a very academic one, for in everything that really constitutes a special malady it is essentially the same. However, its clinical symptoms are somewhat different, and anatomically there is a somewhat more extensive and peculiar change.

Amyotrophic lateral sclerosis, or Charcot's disease, is characterized by progressive paralysis with atrophy, rigidity, and contractures of the limbs.

Etiology.—It is a rarer disease than progressive muscular atrophy, and occurs most often between the ages of thirty-five and fifty, involving the second part of adult life. Rare cases, however, have been reported as occurring in childhood. According to Marie, the female sex is rather more often affected. According to the same author, no definite exciting cause is known. It is not due to syphilis or lead poisoning, nor does it follow the acute infectious diseases. It is therefore considered a disease of involution, *i.e.*, teratological defect, the first and second motor neurons degenerating because of inherently deficient vitality. This state of affairs, however, underlies the other atrophies also.

Symptoms.—The disease begins most often with symptoms referable to the medulla, but it may affect first the arms, and less often the legs. The patient first notices some difficulty in speaking or swallowing. He feels at times a spasmodic drawing of the tongue or stiffness of the cheek or lips. Soon after there appear a weakness and stiffness of the legs and arms. The symptoms progress rather slowly. The speech becomes disturbed; swallowing is difficult; the arms atrophy and become stiff and rigid, producing characteristic deformities. There is great exaggeration of the reflexes; the legs show the presence of ankle clonus; all the arm reflexes are increased, and the jaw is stiff and has a very lively jerk when struck. The patient suffers little from pain. There are no anæsthesia and no sphincter trouble, except in the last stages of the disease. In the course of a year the patient may become quite bedridden, with rigidity and deforming contractures of both arms and legs. In other cases the atrophies and contractures of the arms are not so marked, and the disease shows itself mainly in bulbar symptoms, progressing very much like a case of glosso-labio-laryngeal paraly-

sis, plus a certain amount of rigidity and excessive reflex irritability of the throat and jaws. The course of the disease is variable but usually not long. When it begins and is most marked in the medulla, the duration is shortest. It rarely lasts, in any case, more than two or three years.

Pathological Anatomy.—Post-mortem examinations show a very marked sclerosis involving the direct and crossed pyramidal tracts; also some of the short-fibre systems of the lateral column. The anterior cornual cells are atrophied, as in progressive muscular atrophy. Lesions are also seen at times in the columns of Goll. In fact, the post-mortem findings resemble entirely those of progressive muscular atrophy, except that there is a sharper accentuation of the disease in the lateral tracts. In the medulla the nuclei of the hypoglossal and other motor cranial nerves will be found diseased and the pyramidal tracts also. The lesion of the white columns diminishes in intensity from below up, so that as one gets into the cerebral peduncles very little if any is to be seen. In a few cases, however, the process has been traced to the motor cortex and changes even in that part have been discovered. In a case of my own, which was very closely studied and reported upon by Dr. Jos. Collins, the sclerosis of the motor tracts did not reach above the medulla, and there was no lesion of any moment in the cortical motor cells.

Pathology.—In amyotrophic lateral sclerosis the degenerative process attacks first the terminal fibres and collaterals of the cortical motor neurons. It seems to destroy the tips of the nerve processes, so to speak, without involving the nerve-cell body itself. The next part attacked is the anterior cornual cell. We have therefore the curious and perplexing phenomenon of a disease which attacks the cell body of one neuron and the terminal neuraxon of another neuron just above it. It is difficult to explain this upon the ordinary lines of nerve-cell pathology. Still, we have analogies, perhaps, both in locomotor ataxia and in multiple neuritis.

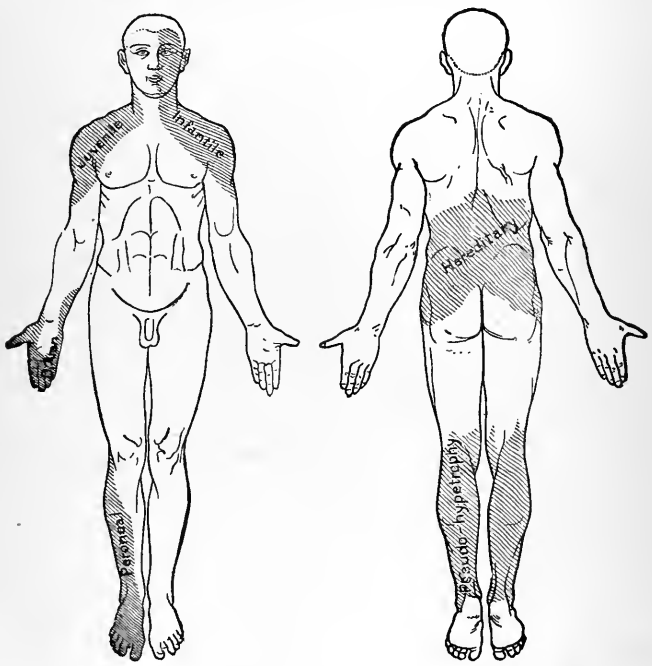
The *diagnosis* of amyotrophic lateral sclerosis must be made from transverse myelitis, multiple sclerosis, and the other forms of progressive muscular atrophy. The diagnosis is based upon the very striking and progressive atrophy associated with exaggerated reflexes, rigidity, and contractures, and without any sensory symptoms or sphincter troubles. The diagnosis from ordinary bulbar palsy depends upon the appearance of stiffness, cramps, exaggerated reflexes, and rigidity displayed by the muscular supply of the facial, the trigeminal, and the glosso-pharyngeal, and the tenth, eleventh and twelfth cranial nerves.

The *prognosis* is invariably bad, but in those types beginning in the legs and arms life may be prolonged a number of years.

The *treatment* is the same as that for progressive muscular atrophy.

THE PROGRESSIVE MUSCULAR DYSTROPHIES.

As I have already stated, there are various forms of progressive muscular atrophy to which the special name of "dystrophy" is



A. B.

FIG. 143.—SHOWING THE PARTS FIRST ATTACKED IN THE DIFFERENT TYPES OF MUSCULAR DYSTROPHY AND MUSCULAR ATROPHY. The shaded parts in A show the place of onset of progressive muscular atrophy of ordinary or Duchenne-Aran type, of leg type, and of types 2 and 3 in text. B shows place of onset of types 1 and (*a*) in text.

given, because they are hereditary in character and because the muscular end of the motor neuron is apparently the first and the most severely attacked. Recent and closer study of the pathology of muscular dystrophy tends to show that the lesion is not in the muscle and terminal of the motor nerves alone, but that the peripheral motor neuron is also to some extent affected. The clinical characteristics of the muscular dystrophies, however, are pretty distinct and are sufficient to justify the separation of them into a different class.

A number of types has been described, the distinctions being based chiefly on the part of the body first affected. These types are not of great importance, but may be enumerated here for convenience (Fig. 143):

1. Pseudo-muscular hypertrophy.

(*a*) Leyden-Möbius or hereditary type, appearing in children, beginning in the back and lower limbs.

2. Erb's juvenile type, or scapulo-humeral type, beginning in childhood or youth, usually in the shoulder girdle or trunk.

3. Landouzy-Dejerine type, or infantile progressive muscular atrophy of Duchenne, or facio-scapulo-humeral type. It resembles the preceding form, with the exception that it involves the face.

4. The peroneal or leg type has been classed with the dystrophies, but is probably of spinal or neuritic origin, and has been described with the atrophies (see page 314).

The essential unity of all these different forms is shown by the fact that cases occur in which pseudohypertrophy takes place in the scapulo-humeral and other types, by the fact that a disease resembling pseudohypertrophic paralysis occurs without any hypertrophy, and by the fact that different types occur in the same family. The unity of the spinal and muscular forms is shown by the same kind of clinical evidence.

At the same time the classical types of dystrophies are very different clinically from the spinal amyotrophies and hence must be separately described. The differences will be shown under the head of diagnosis.

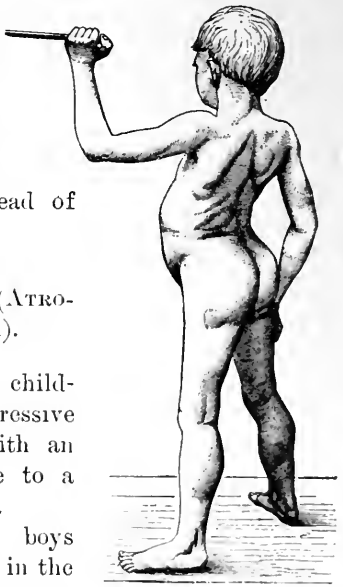


Fig. 144.—Pseudo-Muscular Hypertrophy (Erb).

Pseudo-Muscular Hypertrophy (Atrophia Musculorum Lipomatosa).

This is a disease beginning in childhood and characterized by a progressive weakness of the legs, associated with an apparent muscular hypertrophy due to a deposit of fat in the wasting muscles.

Etiology.—The disease attacks boys much oftener than girls. It begins, in the vast majority of cases, under the age of ten, often at the close of infancy, very

rarely not till after puberty. Heredity is a very important factor (in three-fifths of the cases), the hereditary influence being almost always transmitted by the mother. A psychopathic or neuropathic condition is often found in the ancestry. Neuroses, syphilis, intemperance, consanguinity, are not factors in hereditary causation. Injury and an acute disease sometimes appear to act as exciting causes.

Symptoms.—The first symptom noticed is a weakness in the legs, which shows itself in a peculiar "waddling gait" and a tendency to stumble and fall. A little later (fifth or sixth year) an apparent hypertrophy of the leg muscles, particularly of those of the calves, develops. The extensors of the knee or one of them and the gluteal and lumbar muscles may also be affected. Sometimes the hypertrophy is very great, at other times it is barely noticeable. The affected part has a peculiar, hard, non-elastic feeling to the hand, not like that of normal muscle. In the upper part of the body the hypertrophy oftenest attacks the infraspinatus. The supraspinatus and deltoid may be somewhat involved (Fig. 145). The lower parts of the pectoralis major and latissimus dorsi are also usually atrophied, giving a characteristic appearance to the shoulders. The upper-arm muscles are often slightly wasted, the forearm, neck, and face rarely. The tongue may be hypertrophied.

FIG. 145.—PSEUDO-MUSCULAR HYPERTROPHY, involving legs and shoulders (Curshmann).

Along with the pseudohypertrophy there occurs an atrophy of certain groups of muscles; and after a time the pseudohypertrophy disappears and an atrophy takes its place. In the lower limbs the

muscles most atrophied are the flexors of the hips, then the extensors of the knee and those of the hip. The calf muscles fail before the anterior tibial. The atrophy and consequent weakness of the lower-limb muscles cause great difficulty in going upstairs, the gait becomes more waddling, and the patient loses the power of getting up when lying on the floor. These peculiarities are due chiefly to the weakness in the extensors of the knees, the extensors of the hip, and the flexors of the hip. By reason of the same defects, the child when standing has an antero-posterior curvature of the spine with the concavity backward (lordosis) (Fig. 144). This is due to the weakness of the extensors of the hips, which, acting from the hips, are unable to tilt the pelvis back. On sitting this lordosis disappears, and is replaced often by a curve in the opposite direction due to weakness of the erectors of the spine. There may be some lateral curvature also. In consequence of the weakness and contractures of the leg muscles, there early develops a talipes equinus, and later the legs may become flexed on the hips and the forearms on the arms.

The muscles show no fibrillary twitchings and rarely any degenerative reactions, but there is sometimes a peculiar tetanic contraction with both the faradic and the galvanic current.

The knee jerks and elbow jerks gradually weaken and in time are lost.

There is no pain or other disturbance of sensibility.

The affected parts feel cold and look reddened, as if from deficient vasomotor innervation. The organic spinal centres are not involved. Intelligence is usually good.

Course.—The disease runs a chronic but variable course. Its progress is at first slow; after walking becomes impossible it may cease to progress. It lasts from ten to twenty-five years. In a few cases patients have reached the age of fifty or sixty years, even when the disease began in youth. The earlier the disease begins the more rapidly it extends; the more pronounced the tendency to lipomatosis, the more rapid is the course.

Pathological Anatomy.—The disease, like other forms of dystrophy, is a degenerative atrophy, the process affecting first the muscle fibres and nerve terminals and the connective tissue being secondarily involved. In a simple atrophy of muscle, such as follows disuse, the muscle fibres simply grow smaller and gradually break up and disappear. In degenerative atrophy, the process is accompanied by evidences of irritation, such as swelling of the muscle fibre, proliferation of muscle nuclei, splitting of the fibre longitudinally, and connective-tissue proliferation. All these phe-

nomena are seen in the pathological process which takes place in the dystrophic muscles. All the varied changes may be noted in the same muscle. In the early stages there is a true hypertrophy of some of the fibres, a condition thought to be characteristic of the muscular dystrophies in distinction from the spinal atrophies. Besides swelling and hypertrophy of fibres, one sees atrophy of the fibres; the bundles are rounded; there are increase of muscle nuclei,

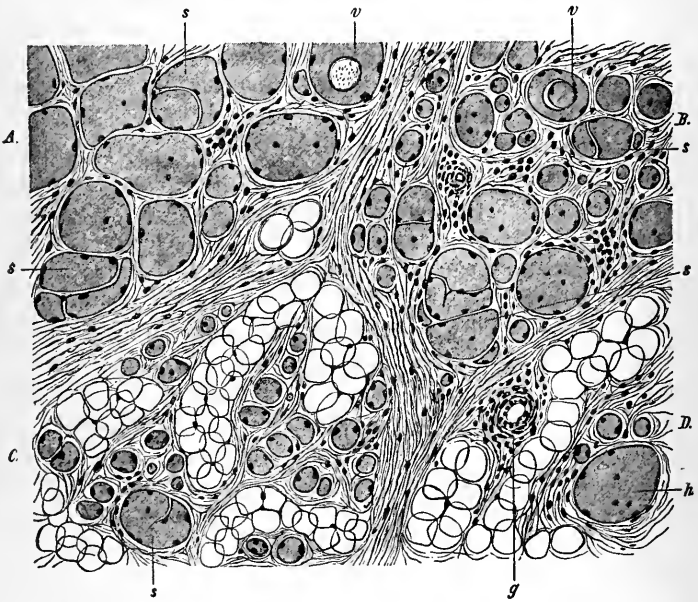


FIG. 146.—PARTIALLY DIAGRAMMATIC, SHOWING: *A*, hypertrophied fibres; *B*, mixture of hypertrophy and atrophy; *C*, *D*, atrophy and fatty deposit; *v*, vacuolization; *s*, splitting; *h*, hypertrophy of fibres; *g*, thickened blood-vessel (Erb).

splitting of fibres, vacuolization, and a tendency to break up into fibrillæ (Erb). The connective tissue at first shows evidence of irritation and proliferation. Finally, as the muscular atrophy progresses, connective tissue increases and takes its place, until a dense, hard myosclerosis results (Fig. 146). In some parts there is deposit of fat in the connective-tissue cells, and this may increase until an extensive lipomatosis exists. In the later stage of the disease the fat deposits are absorbed and there are only atrophied muscle and connective tissue. The nerves and spinal cord are usually normal; when changes are found they are secondary to the muscular disease.

The process is then, first, hypertrophy of muscle fibre and

increase of muscle nuclei, swelling and rounding of fibres, and splitting of the same; then increase of connective tissue, with corresponding atrophy of muscle and deposit of fat.

The process is a primary degeneration due to an inherent nutritional weakness of the muscle. In a measure it is true that those muscles embryologically latest developed are earliest attacked.

The JUVENILE DYSTROPHY OF ERB, or scapulo-humeral form of dystrophy, begins in childhood or early youth, a little later than pseudohypertrophy. The shoulder girdle is first affected, later the arm. The forearm and legs are attacked very late. Part of the pectorals, part of the trapezii, latissimus dorsi, rhomboid, upper-arm muscles, and supinators are affected, while the supra- and infraspinati and forearm and hand usually escape. There may be true and false muscular hypertrophy. There are no fibrillary contractions or degenerative reactions (Fig. 147).

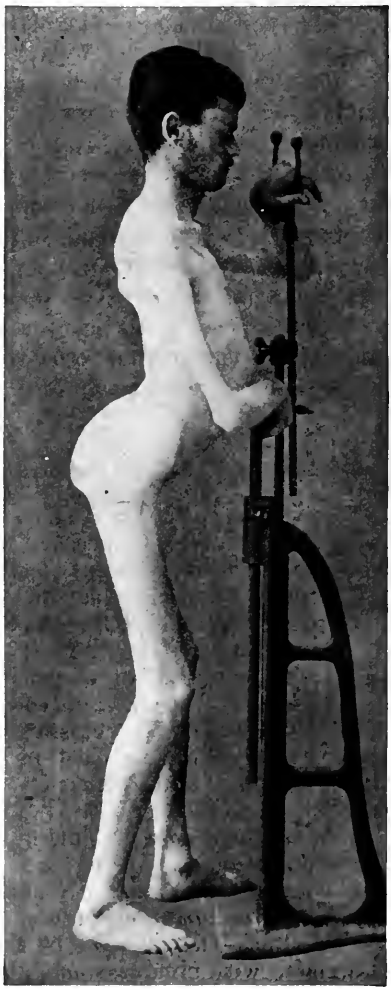


FIG. 147.—JUVENILE TYPE OF PROGRESSIVE MUSCULAR DYSTROPHY; sixth year of disease.

The FACIO-SCAPULO-HUMERAL FORM, or infantile progressive muscular atrophy, begins in early childhood (third or fourth year) usually, but may develop late. The atrophy attacks first the face, giving a characteristic appearance known as the "myopathic face." There is a weakness of the oral muscle, which causes the lips to protrude and produces a symptom called the "tapir mouth." The atrophy respects the eye muscles as well as those of mastication and deglutition. It extends to the shoulders

and arms next, then it pursues the ordinary course of the dystrophies.

Prognosis.—The patient never recovers, but the disease sometimes comes to a standstill and there may even be some improvement, especially in cases beginning late.

Treatment.—The prophylaxis is important. It consists in preventing the marriage of women belonging to dystrophic families; if a dystrophy has developed in one child, it would be unwise to take the risk of bringing others into the world. Or if children are already born, they should receive the most careful nourishment, outdoor life should be secured, and the dangers from trauma and the infective diseases be prevented. Infants should not be suckled by the mother if she belongs to the dystrophic family.

The moderate use of massage and gymnastics is very important and useful. All kinds of tonic measures are indicated, such as cold baths, good nourishment, arsenic, strychnine, and phosphorus and fats. Tenotomy and other orthopædic measures may be useful in the later stages. Feeding with thymus gland may be tried.

Summary of the Hereditary or Family Nervous Diseases.

The student may well be confused by the large number of so-called family nervous diseases which modern neurology has discovered and differentiated. The practical importance of them all is, perhaps, slight, for they are extremely rare, yet it is necessary that they be recognized and properly distinguished, for the prognosis and degree of suffering differ very much in different cases. They are all characterized by the fact that they are found in different generations and in different collateral branches of a given family, and that they are not necessarily or often passed on directly from one parent to another. The list which I append may not be entirely complete at the time of publication of this book, but it is ample.

Hereditary Chorea.—This is really a kind of hereditary paresis or brain softening. It does not develop until adult life, as a rule, and patients with it may live until middle age.

Hereditary amaurotic idiocy is a family disease first described by Dr. Sachs, characterized by lack of development of the brain and associated with blindness and a peculiar degeneration of the optic nerves.

Hereditary Cerebral Diplegia.—This is a family disease in which children between the ages of one and five develop spastic paralysis and sometimes imbecility.

Hereditary Hemiplegia.—This is a family disease which has been referred to by Hoffmann and of which I have seen two cases in my clinic. The children are born hemiplegic and there is atrophy

of the hemiplegic side, but in my cases there was no mental defect or epilepsy.

Hereditary cerebellar ataxia is a disease allied to Friedreich's ataxia, but developing somewhat later, *i.e.*, about the time of puberty.

Hereditary spinal ataxia, or Friedreich's disease, is fully described elsewhere.

Hereditary ataxic paraplegia is a disease developing at about the same time and with some of the same symptoms as cerebellar ataxia.

Hereditary spastic spinal paralysis is a disease which as far as is known develops before the fifth or sixth year of life, involves only the lower extremities, and is compatible somewhat with a long life. It resembles ataxic paraplegia.

Hereditary progressive spinal muscular atrophy is a family disease, developing in the first year of life, affecting first the legs, and gradually ascending and running a rapid course, the patient dying in three or four years (Hoffmann).

The hereditary progressive dystrophies include a large number of progressive diseases affecting primarily the muscles and the muscular ends of the peripheral motor neurons. Many different types of this disease are described in accordance with the muscles which are first attacked.

Arthritic Muscular Atrophy.

In inflammation of joints the muscles moving them are affected by a simple atrophy which is called arthritic.

Etiology.—Rheumatic arthritis is the commonest cause.

Symptoms.—The shoulder-girdle muscles are oftenest affected. Whatever the joint, it is the extensor muscles which are first and most attacked; the muscles above the joint are more susceptible to the atrophy than those below. The atrophy is rather rapid in the first few weeks and then becomes slower. The muscles affected waste throughout their whole length. They show no fibrillary contraction and no degenerative electrical reaction. There is often an increased irritability, so that an exaggerated tendon reflex or even clonus may be produced. There is no pain or tenderness or anæsthesia in the muscles.

Pathology.—The anatomical change is a simple atrophy and shrinking of the muscle fibres, with some increase in muscle nuclei, little vacuolation, no swelling or splitting of fibres (Darkschewitch —Fig. 148). There is some increase in interstitial tissue, but this is slight. The nerves and spinal cord are normal.

The atrophy is probably due to reflex action. It is a reflex trophoneurosis. Even a neuralgic joint may cause an atrophy.

The *prognosis* is good. If the arthritis gets well the muscles are also restored.

The *treatment* consists of electricity and gentle massage and exercise. Internal treatment must be directed to the arthritis.

Occupation Muscular Atrophies.

As a result of constant overuse muscles sometimes atrophy. This applies especially to the smaller muscles of the hand. Thus

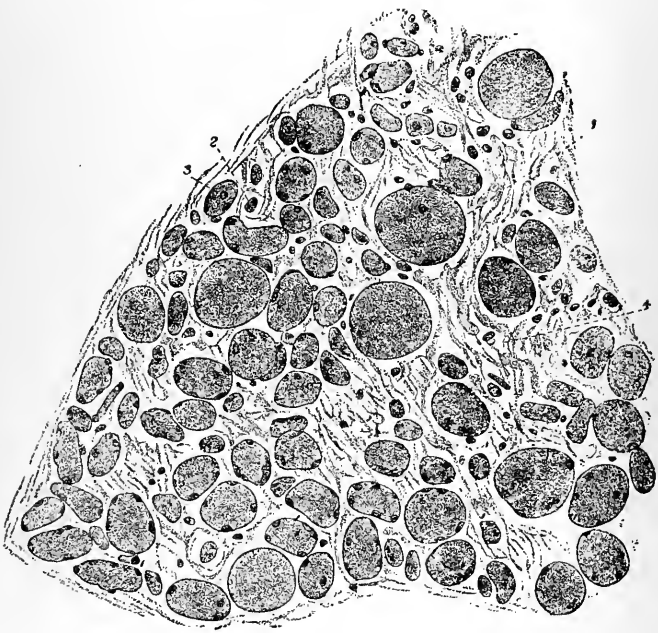


Fig. 148.—Showing Changes in Arthritic Muscular Atrophy. 1, Normal fibre; 2, atrophied fibre; 3, vacuolated fibre; 4, connective-tissue proliferation (Darkschewitch).

there occurs an atrophy of the thenar eminence in lapidaries and in persons who constantly use this group of small muscles.

Typical atrophies of this kind may be seen in persons who run elevators and who have constantly to grasp the rope in one hand. The biceps sometimes wastes in smiths and the calf muscles in ballet dancers. In most cases this occupation atrophy of muscles reaches a certain stage and stops. If the patient is given rest, recovery takes place. This is especially true if the patient is young and in vigorous health. In other instances, the simple occupation atrophy will

actually pass over into progressive muscular atrophy. The condition is distinguished from a true progressive muscular atrophy mainly by the fact that the patient has none of the aching pains and vaso-motor symptoms, such as sweating in the arm, and very little if any fibrillary tremor. The electrical reactions are the same as in spinal atrophies. In ordinary occupation atrophy the seat of the disease is probably in the muscle itself. The treatment is rest and tonics, the careful application of electricity, and hypodermic injections of strychnine.

CHAPTER XVI.

TUMORS AND CAVITIES OF THE SPINAL CORD.

Spinal Tumors.

Etiology.—Tumors rarely occur in the spinal cord. The commonest age is thirty to fifty; tubercle occurs earlier (fifteen to thirty-five—Herter) and lipoma is congenital. Males are more subject than females. Tuberculosis, syphilis, and cancer predispose to the disease. Injuries and exposures appear sometimes to excite the growth of spinal neoplasms.

Symptoms.—These vary with the location, character, size, and rate of the growth of the tumor. No definite clinical picture can be drawn. The symptoms are such as result from a foreign body slowly and progressively irritating and destroying the roots and substance of the spinal cord. Pain appears early and is very constant, continuous, and severe. It is generally referred to nerves running out from the cord in the region of the tumor; a girdle sensation is felt. Numbness, hyperæsthesia, and later anæsthesia occur. Tenderness over the spine and rigidity are not very frequent. The sensory symptoms are usually more on one side, but may become bilateral. Spasm, contracture, and exaggerated reflexes usually soon develop, involving one or both legs or an arm and a leg. Later paraplegia, atrophy, loss of control of the bladder and rectum, and bedsores follow, and death ensues from exhaustion.

When the disease is cervical the four extremities and trunk muscles may be gradually involved, and there are rigidity of the neck and optic neuritis. If lower down, there develops a hemiparaplegia, later a complete paraplegia, usually with exaggerated reflexes. If the tumor is in the lumbar region the reflexes are sooner lost and the sphincters early involved.

A rather frequent type of symptoms caused by spinal tumors is that known as a *Brown-Séquard paralysis* or hemiparaplegia. In a typical case of this kind there are paralysis of motion and muscle sense on the side of the lesion, paralysis of cutaneous sensation, especially of pain and of temperature on the opposite side. On the side of the lesion the temperature may be slightly raised; there is often hyperæsthesia, and reflex action is increased. There may be a

band of anæsthesia at the level of the lesion and on the same side (Figs. 149, 150).

The symptoms vary according as the tumor is outside or inside the dura. The common extradural forms are lipoma, cancer, gumma, and sarcoma. The greater amount of motor and sensory irritation, the evidence of some vertebral disease, existence of malignant tumor elsewhere, the absence usually of hemiparaplegia, characterize extramedullary tumors. The common forms of intradural or medullary tumor are glioma and tubercle. In these cases pain and spasm and rigidity are less common in the early stage; hemiparaplegia is more common. A secondary myelitis sometimes develops.

The duration of the disease ranges from three to five years, the average being two or three years.

Pathology and Pathological Anatomy.—All forms of tumor occur, but the commonest are gliomata and sarcomata, and after this fibromata, myxomata, gummata, and tubercles. Cancer is rare; echinococcus and cysticercus are the only parasitic tumors found. Most new growths start from the membranes, the next largest numbers from the cord, and fewest from the vertebræ.

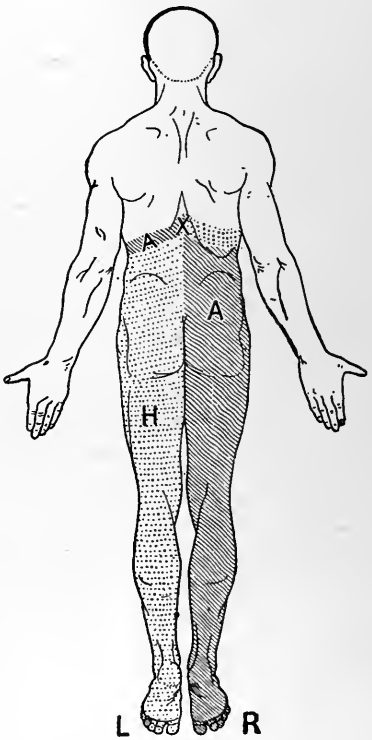


FIG. 149.—SHOWING THE CONDITION IN A BROWN-SÉQUARD PARALYSIS DUE TO A TUMOR GROWING IN THE LEFT SIDE OF THE SPINAL CORD. On the left side, hyperæsthesia, ataxia, paralysis, exaggerated reflexes. At the upper limit is a band of anæsthesia. On right side, anæsthesia.

The meningeal tumors are mostly sarcomata and their various modifications, fibroma, enchondroma, carcinoma, and lipoma. The myelonic or intraspinal tumors are commonly gliomata, sarcomata, tubercle, and syphilomata.

Spinal tumors are small in size, ranging from one-fifth to one and one-fifth inches (one-half to three centimetres) in diameter. The glioma may diffuse itself for a long distance through the centre of the cord, forming cavities (syringo-myelia). The sarcomata may

likewise be irregularly spread along the surface of the cord. Spinal tumors are usually single, but fibromata, the parasites, and sarcomata may be multiple.

The favorite locations for spinal tumors are just below the mid-cervical, the upper and the lower dorsal regions. The secondary changes produced by tumors are softening, hemorrhages, secondary degenerations, and inflammatory reaction. The origin and histology of spinal tumors do not differ from that of tumors elsewhere.

The Diagnosis.—The disease has to be distinguished from vertebral caries, transverse myelitis, and hypertrophic pachymeningitis.

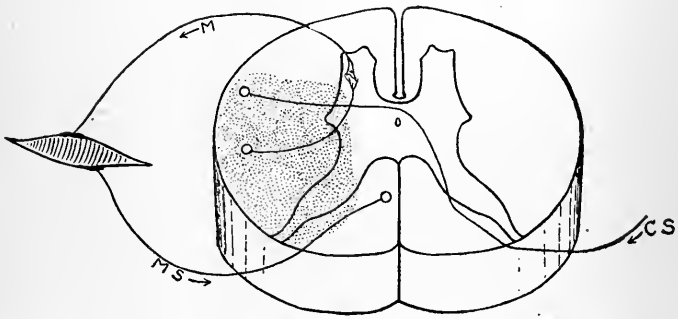


FIG. 150.—SHOWING THE MECHANISM OF THE PRODUCTION OF A BROWN-SÉQUARD PARALYSIS. The shaded part on the left represents the lesion. This involves the pyramidal tract and motor fibres *M*, causing paralysis and spasm on the same side; also the muscle sense nerves *M S* on the same side and the cutaneous sensory nerves *C S* on the opposite side.

The points to be noted as regards caries are the absence of an external tumor or kyphosis; the small degree of tenderness and rigidity, the age, and absence of tubercular diathesis. The progressive course, beginning with pain, followed by motor and then sensory paralysis, and the localization of the symptoms exclude myelitis. The differentiation from hypertrophic pachymeningitis is often very difficult.

The character of the tumor cannot often be determined certainly. The probabilities are in favor of sarcoma or glioma, especially in middle life. Syphiloma may be suspected from the history and results of treatment. Tubercle is very rare.

The *prognosis* is bad except in syphiloma, and even here it may be serious. Tubercle may perhaps cease to grow. Surgical interference now saves the life of some patients.

Treatment.—In syphilitic tumors appropriate remedies may do good and should be vigorously used. In tubercle, tonic treatment, iodine, and cod-liver oil may be of service. In gliomata and sarcomata, nitrate of silver and arsenic may be tried. In other forms, symptomatic treatment is all that can be recommended medi-

cally. In all cases of spinal tumor surgical interference should be considered; extradural tumors, if taken early, can be removed with great benefit. Even in medullary tumors enucleation may do some good in the early stage. Exploratory operations are justifiable if there is no question as to the diagnosis of tumor. The mortality from such operation in capable hands is very low, though it is more dangerous than trephining the skull.

FIG. 151.—SARCOMA OF SPINAL CORD, MID-CERVICAL REGION.

CAVITIES IN THE SPINAL CORD.

The cavities of the spinal cord are known as:

1. Hydromyelia.
2. Myelitic cavities.
3. Syringomyelia.

There may be various combinations of those processes, the most common being a combination of hydromyelia and syringomyelia.

HYDROMYELIA.

This is a dilatation of the central canal of the cord, the cavity being filled with fluid. The condition may be associated with hydrocephalus or with spina bifida, or it may be independent of these conditions. The dilatation may be cystic and irregular, or, as is more usual, extend throughout the cord. The dilatation extends more posteriorly, because the posterior columns are formed latest. The abnormality may be slight and give rise to no symptoms. Or a pathological process like a gliomatosis may develop upon it.

MYELITIC AND HEMORRHAGIC CAVITIES.

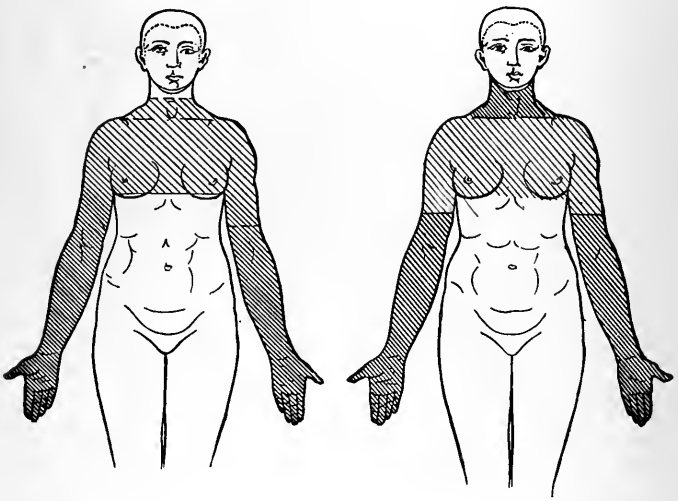
Such cavities may be formed in the cord by a central excavating myelitis or by small hemorrhages. These cavities are usually small, irregularly distributed, and are lined with connective tissue. They may be formed in rare cases in connection with hydromyelia or neoplasms.

Syringomyelia.

Syringomyelia is a disease of the spinal cord characterized by a development of gliomatous tissue in the central parts, with formation of cavities. Clinically the disease is usually characterized by peculiar disturbances of sensation and nutrition. In many cases, however, the symptoms are atypical and the disease cannot be recognized during life.

The description here given corresponds to the classic manifestation of the disease, and it is intended to refer to syringomyelia produced by gliomatosis only.

Etiology.—The disease is rare, but over two hundred cases have now been reported. It is more frequent than amyotrophic lateral



1. 2.

Fig. 152.—Distribution of Cutaneous Anæsthesia in Syringomyelia. 1 shows area of analgesia, 2 shows that of thermo-anæsthesia. The darker shades show where there is anæsthesia to pain and temperature, the tactile sense being unimpaired.

sclerosis and less frequent than multiple sclerosis. It occurs oftener in men than in women, and develops early in life, between the ages of fifteen and twenty-five.

It occurs especially in persons who follow manual occupations, such as butchers, tailors, etc. Traumatism, pregnancy, and infectious diseases seem occasionally to give rise to it. Heredity, syphilis, and alcohol are not causal factors.

Symptoms.—The disease begins insidiously with some aching pains in the neck and arms and paræsthesia of the hands. There is soon a muscular atrophy of the hands resembling spinal progressive muscular atrophy, with perhaps anæsthesia. As the disease pro-

gresses the weakness and atrophy of the hand muscles become more noticeable and gradually extend toward the trunk. The atrophy comes on in both extremities at about the same time. Fibrillary contractions and partial degeneration reaction may be observed. Cutaneous anæsthesia of the affected hand and arm to temperature and pain, but not to touch, takes place; and this is so marked as to be almost pathognomonic of the disease (Fig. 152).

The legs do not become affected until late, and then generally show a spastic paraplegia. The throat and face are rarely involved. There is a scoliosis of the spine, generally in the dorsolumbar region (Fig. 153).

Vasomotor, secretory, and trophic symptoms are prominent. The hands may be œdematous or red and congested. Sweating or dryness of the skin may occur. Eruptions appear on the skin, such as bullæ, herpes, and eczema. Painless whitlows attack the fingers and may destroy the terminal phalanges. Erosions and ulcerations also occur. The nails become dry, brittle, and drop off. Arthropathies and spontaneous fractures have been observed. The pupils may be unequal and the bulb retracted.

Late in the disease symptoms of involvement of the medulla develop. At this time also the bladder, rectum, and genital centres are attacked. The disease progresses slowly for years, with remissions of various degree.

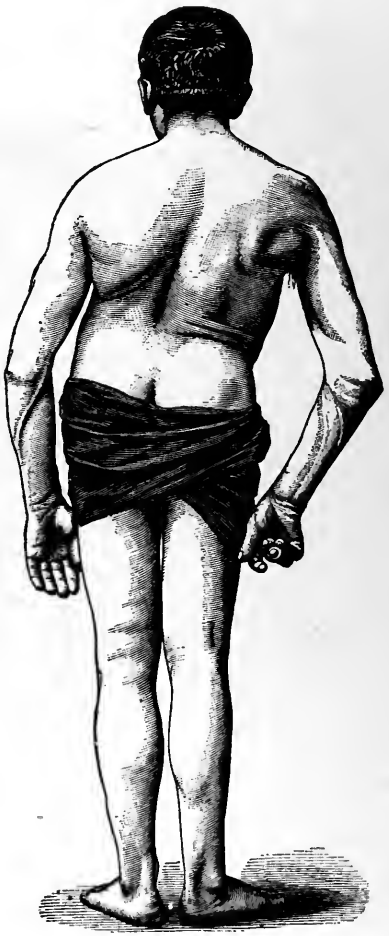


FIG. 153.—SHOWING CURVATURE OF SPINE IN SYRINGOMYELIA (Erb).

The cardinal symptoms are a progressive muscular atrophy, with a peculiar partial anæsthesia, trophic disturbances, and scoliosis.

Pathological Anatomy.—The seat of the disease is the substance of the cord. On opening it by transverse sections one finds one or more cavities extending in various degrees up and down. The most frequent primary site is the cervical swelling. From here it usually

passes down and may reach the whole length of the cord. It also extends upward and may involve the medulla and the nuclei of the cranial nerves. The cavities are of irregular shape, small size, and

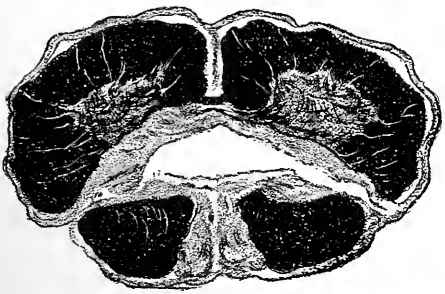


FIG. 154.

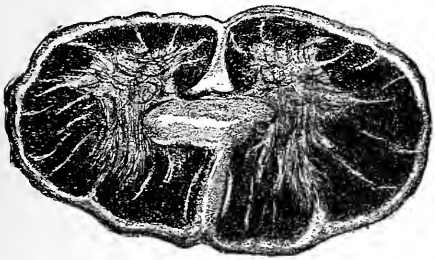


FIG. 155.

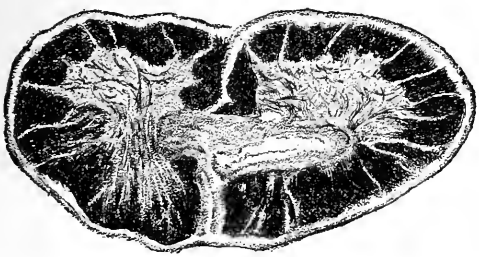


FIG. 156.

FIGS. 154-156.—SECTIONS OF SPINAL CORD AT DIFFERENT LEVELS IN SYRINGOMYELIA (Bruhl).

FIG. 157.—SYRINGOMYELIA, showing cavity with gliomatous tissue around it, and ascending and descending degeneration.

filled with a liquid like the cerebro-spinal fluid. They are situated oftenest posterior to the commissure and involve one or both posterior horns, but they may be so extensive as to involve almost the whole of the centre of the cord at some levels. The walls are usually lined by a membrane and surrounded by a gliomatous tissue (Figs. 154–157).

This membrane is composed of a rather dense gliomatous tissue.

It may be absent in some parts. The *glia* cells are in various stages of development and degeneration. In parts of the cord the new growth may form a large and solid mass occupying most of the centre of the cord. Small hemorrhages and foci of myelitis may be present. In some cases there is evidence of a dilated central canal, with neuroglia hyperplasia of the walls and a gliomatous infiltration about this. The epithelium of the central canal may form part of the wall of the cavity.

Pathology.—At about the sixth week of fœtal life the central canal of the cord is large, diamond shaped, and reaches nearly to the anterior and posterior surfaces of the cord. This cavity gradually contracts and unites in the middle, the anterior part forming the central canal and the posterior part the posterior septum. The posterior part may, through some embryological defect, fail to close, and a congenital cavity may be left. About this a glioma may develop, and we have syringomyelia. The central canal may remain imperfectly contracted, and a syringomyelia may develop in connection with it. Probably most cases of syringomyelia are developed on the basis of an embryonic defect.

Gliomata were formerly thought to be practically identical with sarcomata. It is believed now, however, that they are of epiblastic origin, and like nerve cells are modifications of epithelial tissue. This tissue in its normal state is called neuroglia. It is a nervous substance. It is composed of small cells, round or of irregular shape, with a large nucleus and fine fibrillary prolongations. In glioma these cells are relatively much more numerous, while the fibrillary network is less conspicuous. There is considerable variation in the relative richness of cells and fibres, however. When the former are very frequent the term gliosarcoma has been wrongly given to the tumor. The glioma is penetrated by small blood-vessels whose walls are often diseased, so that minute hemorrhages occur and the glioma becomes stained and pigmented. In other cases it is gray or yellowish in color.

The rich cellular proliferation in gliomata has suggested an analogy in its growth to that of inflammation, and the term gliosis is used as analogous for neuroglia to sclerosis of connective tissue. Gliosis differs from sclerosis, however, in the fact that in the latter process the multiplication of fibres dominates, while in the former it is the cells; besides this, in gliosis there is a tendency to softening and formation of cavities, and all nerve fibres disappear. In sclerosis some nerve fibres remain, and one observes the presence of granular and amyloid bodies.

Peculiar Types.—1. The disease may be latent, giving rise to very few symptoms or to none that are characteristic.

2. There may be a period of irritation and pain in the extremities followed by paraplegia, with few sensory troubles, the course suggesting a chronic transverse myelitis or a Brown-Séquard paralysis.

3. There is a type in which bulbar symptoms develop early, but

differing from ordinary bulbar paralysis in the involvement of the trigeminus and other cranial nerves not commonly attacked.

4. There is a form characterized by a rather rapid ascending paralysis.

None of these types can ordinarily be recognized during life.

5. There is a type characterized by the symptoms of muscular atrophy with analgesia and felons (Morvan's disease). In this type there is probably a complicating neuritis. Some assert that all cases of Morvan's disease are cases of syringomyelia, but this is not proven.

Diagnosis.—The disease is distinguished in its classical form by (1) its beginning at the *period of adolescence*, (2) by the progressive muscular atrophy combined with the peculiar dissociated disturbances of sensibility, (3) by the trophic disturbances and scoliosis.

It has to be distinguished from progressive muscular atrophy and dystrophy, and amyotrophic lateral sclerosis, hypertrophic cervical pachymeningitis, chronic transverse myelitis, Morvan's disease, and anæsthetic leprosy.

The sensory and trophic disorders and scoliosis enable one to distinguish it from progressive muscular atrophy. In leprosy the dissociation of the sensory symptoms is not present, and the anæsthesia is distributed along the course of the nerves or in sharply circumscribed plaques. In some cases the peculiar tubercular disease of the skin and the history of the case make the diagnosis easy. In leprosy, also, there is a perineuritis, and the enlarged inflamed nerves may be felt. Portions of the skin may be excised and examined for the leprosy bacillus. As regards the differentiation from Morvan's disease, this cannot often be done. Still whitlows are rare in ordinary forms of syringomyelia. Morvan's disease begins in one hand and slowly extends, with remissions, to the other. Usually there is loss of tactile as well as thermic and pain sense.

The prognosis so far as life is concerned is bad; but the disease has often a long course, ranging from five to twenty years, and periods occur in which the progress of the disease seems arrested and improvement occurs.

Treatment.—It is not impossible that we may find some drug which acts specifically on gliomatous tissue, checking its growth. At present we know of only two things which may possibly do this: nitrate of silver and arsenic. These drugs should be given; and for the rest, tonic and symptomatic treatment is indicated.

Functional Disorders of the Spinal Cord.

The functional disorders of the spinal cord include only conditions of irritation and depression, to which the names of spinal irritation and spinal exhaustion or spinal neurasthenia are given. The symptoms of these states are sufficiently described under the head of neurasthenia.

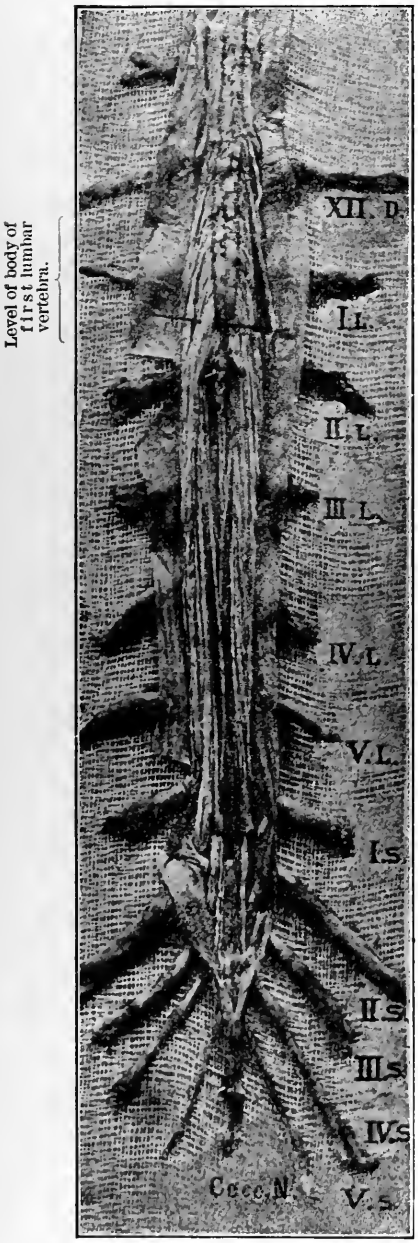


FIG. 158.—SHOWING CAUDA EQUINA, NATURAL SIZE (Müller).

THE RECOGNITION OF DISEASES OF THE CAUDA EQUINA.

Anatomy.—The cauda equina is made up of five lumbar, five sacral, and one coccygeal nerve roots. They lie in the dura mater forming a thick bundle and extending down the vertebral canal for 14 cm. They are still distinct motor and sensory roots, and do not unite till they have passed out of the dura. The cauda begins at the lower tip of the cord, at the level of the lower edge of the second lumbar vertebra. The term conus is applied to the lower end of the cord and includes the parts below the second sacral segment. The cord here becomes much smaller, loses some of its distinctive microscopical structure, and the anterior root fibres are smaller and less numerous than the posterior or sensory.

The arrangement of the segments and nerves is shown in Figs. 158 and 159. The arrangement of the visceral centres is given by Müller as follows: Second sacral, erection centre; third sacral, ejaculation centre; fourth sacral, bladder (detrusor) centre; fifth sacral, sphincter-ani centre. The distribution of the sensory nerves is shown in the Figs. 160 and 161.

Symptoms and Diagnosis.—The diagnosis of cauda lesions

involves differentiation of (1) lesions of the lower end of the spinal cord; (2) cauda lesions due to compression; (3) those due to destruction; (4) lesions of the peripheral nerves.

1. Lesions of the lower end of the cord usually come on rapidly, *i.e.*, in a few days (myelitis, softening, hemorrhage); there is little

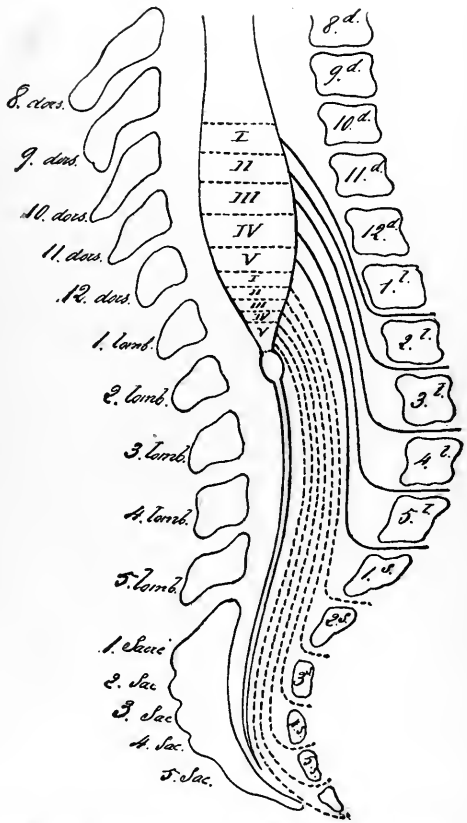


FIG. 159.—SHOWING ARRANGEMENT OF SEGMENTS AND NERVES OF CAUDA EQUINA (Peterson).

pain or sensory irritation, and later there is dissociation of sensations. Fibrillary contractions and involuntary twitchings of the leg muscles occur. Paralysis rapidly appears, involving the lower limbs in accordance with the segmental distribution of the nerves. It is flaccid, and is followed by atrophy. The visceral centres are involved. If the conus is not implicated the paralysis does not seriously involve these centres, nor the muscles of the

pelvic girdle. The motor disturbances are more conspicuous and troublesome symptoms than are the sensory disturbances. The cord being small and destroyed, not much improvement takes place.

2. In disease of the cauda, since it is usually a tumor, the symptoms come on slowly; in injury, however, the onset is sudden. There

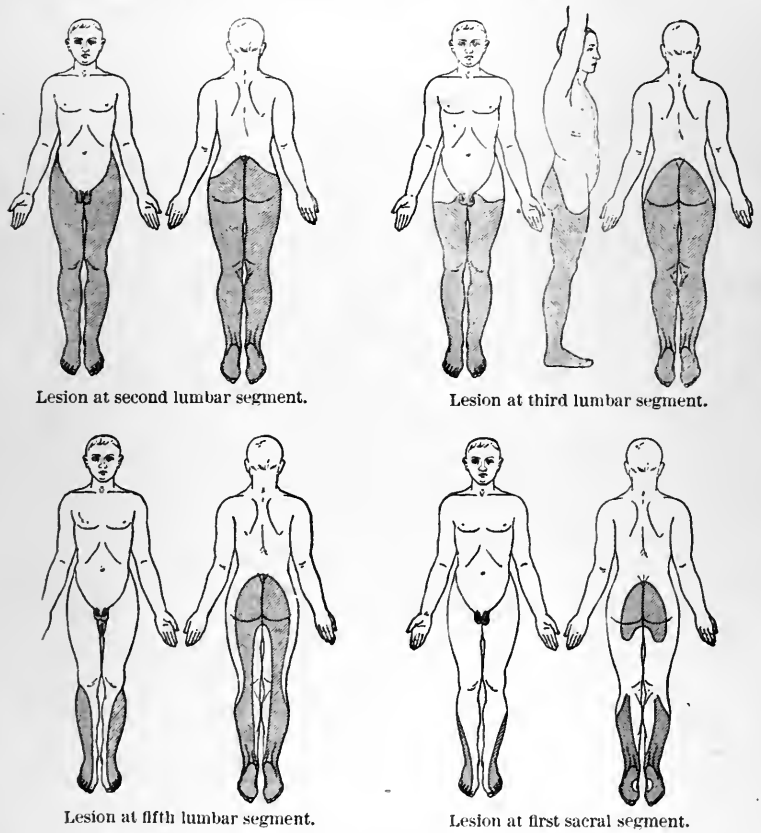


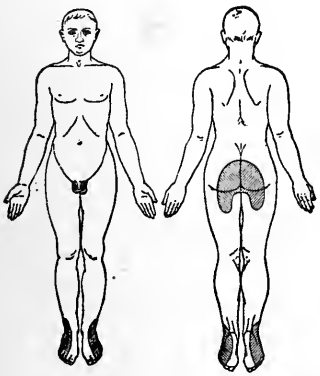
FIG. 160.—LESIONS AT DIFFERENT LEVELS OF THE LUMBAR AND SACRAL CORD, SHOWING AREAS OF ANÆSTHESIA (Müller).

is often severe pain felt in the bladder and distribution of the sciatic nerves, and usually bilaterally. There follows after a time anæsthesia in the course of the sciatic nerves. There is little motor irritation, and paralysis follows slowly, accompanied with pain, the sensory symptoms being all along in the foreground. The sexual, bladder, and rectal centres are later paralyzed. The course is progressive unless there is effective operation or medical interference.

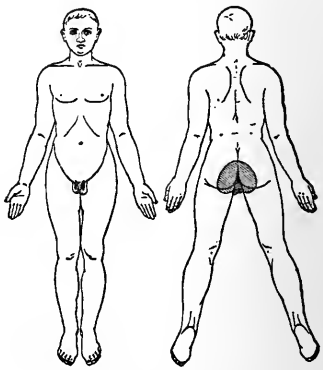
3. The symptoms in compression of the cauda without destruc-

tion are much the same as the above, but there is less motor disturbance, and there may be no involvement of the visceral centres.

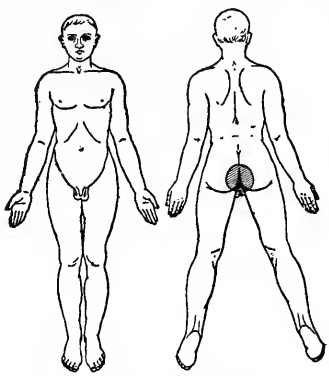
4. In lesions of the peripheral nerves, the trouble (usually neuritis or injury) comes on rather rapidly. There are sciatic pains, tender points, the lesion may be only unilateral, the pains are not



Lesion at second sacral segment.



Lesion at third sacral segment.



Lesion at fourth sacral segment.

FIG. 161.—LESIONS AT DIFFERENT LEVELS OF THE SACRAL CORD, SHOWING AREAS OF ANÆSTHESIA (Müller).

so severe, and there is no marked anæsthesia. There is little or no paralysis in sciatica, but it may occur in neuritis. There is no paralysis of the visceral centres; the sensory and motor symptoms go together, the sensory slightly predominating; there is often a history of sciatica and alcoholism, or injury. Examination may disclose the presence of a tumor or of some disease affecting directly the sciatic plexus, and the prognosis is favorable.

CHAPTER XVII.

ANATOMY AND PHYSIOLOGY OF THE BRAIN.

Anatomy.—The nervous system is developed from a hollow tube formed by a folding of the epiblast. The brain or encephalon grows out from its anterior part. This swells into three cavities called the anterior, middle, and posterior cerebral vesicles. From the

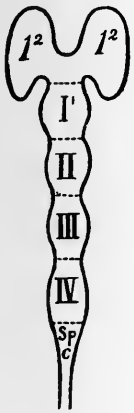


FIG. 162.

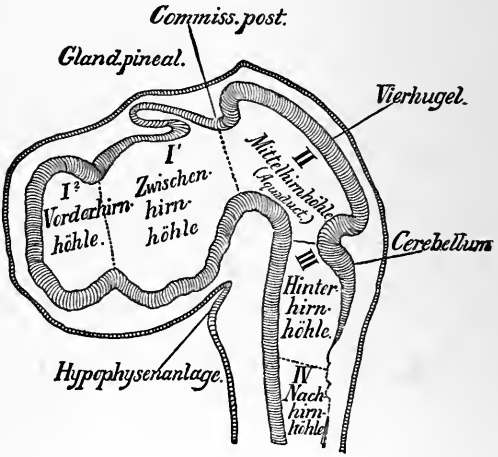


FIG. 163.

FIG. 162.—THE CEREBRAL VESICLES.

FIG. 163.—FURTHER DEVELOPMENT OF VESICLES.—I^2, Fore-brain or telencephalon: I^1, 'tween-brain or diencephalon; *II*, mid-brain or mesencephalon; *III*, isthmus and metencephalon or hind-brain. The isthmus is not indicated separately in the above figure. *IV*, after-brain or myelencephalon or medulla oblongata (Edinger).

anterior a secondary vesicle develops; the posterior divides into two; so that eventually there are five vesicles. Out of them the different parts of the brain are formed.

1. From the anterior vesicle there grow the cerebral hemispheres, the corpus callosum and anterior commissure, fornix, corpus striatum, and olfactory lobes. It includes also the anterior part of the region lying under the thalamus in which are the optic chiasm and pituitary body. These structures form the fore-brain or telencephalon.

2. From the posterior part of the primary vesicle come the thalamus, pineal gland (or epithalamus), geniculate bodies (or metathalamus), and some structures lying under the thalamus, viz., the

corpora mamillaria, and Luys' body. These parts form the 'tween-brain or diencephalon.

3. From the middle cerebral vesicle there grow the corpora quadrigemina and cerebral peduncles, with the red nuclei and substantia nigra. They form the mid-brain or mesencephalon.

4. From the fourth vesicle, which is a secondary vesicle de-

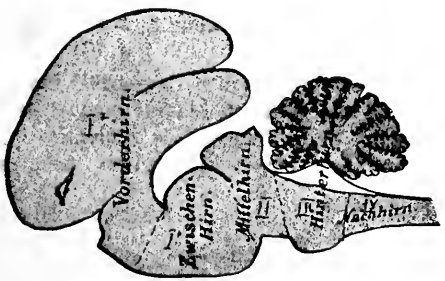


FIG. 164.—STILL FURTHER DEVELOPMENT OF VESICLES. The cerebrum is being formed out of the first vesicle I² (Edinger).

veloped from the third primary, come the "isthmus" (which includes the superior cerebellar peduncles and valve of Vieussens) and the "after-brain," composed of the cerebellum, its middle peduncles, and the pons. This part is also called the metencephalon.

5. The fifth vesicle (also a development from the third) forms the medulla oblongata, or after-brain, or myelencephalon.

The development of these parts is shown in the accompanying diagrams. In man the fore-brain is enormously developed, the 'tween-brain moderately developed, the olfactory lobes are atrophic,

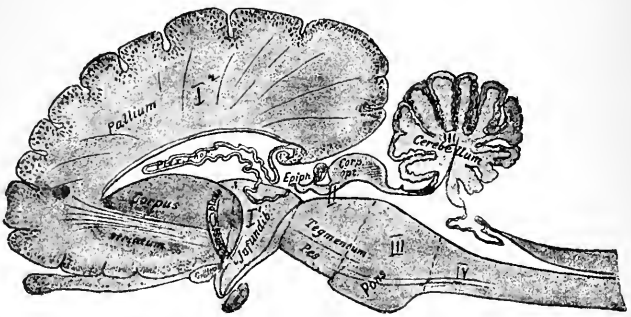


FIG. 165.—FURTHER DEVELOPMENT. The dotted lines show the original subdivision into vesicles (Edinger).

the mid-brain is almost rudimentary, the hind-brain well developed, the after-brain relatively not much developed. The divisions above given in detail seem somewhat academic and impractical as applied to adult human brains, but the main features are not so; indeed

they are quite essential to the understanding of modern anatomy. The following simplification is sufficient for the student:

Fore-brain or telencephalon.	Cerebral hemispheres and corpora striata
'Tween-brain or diencephalon.	Optic thalamus and geniculate bodies.
Mid brain or mesencephalon.	Cerebral peduncles and corpora quadrigemina.
Isthmus and hind-brain or metencephalon.	Cerebellum and its peduncles. Pons.
After-brain or myelencephalon.	Medulla.

In the process of development of the brain, the neural canal becomes variously enlarged and constricted, until the ventricles of the brain, the foramina of Monroe, and the aqueduct of Sylvius are formed. The ventricles are the two lateral, the third, fourth, and fifth. The foramina of Monroe connect the lateral and third ventricles; the aqueduct of Sylvius connects the third and fourth ventricles.

Along the mesial or inner surface of each optic thalamus runs a groove known as the *fissure of Monroe*. This, according to Minot, may be traced along the sides of the aqueduct of Sylvius and fourth ventricle into the spinal cord, where it is identical with the line of division that in embryonic life separates the dorsal from the ventral parts of the cord, forming the dorsal and ventral zones of His. Minot regards this line of demarcation as having great morphological importance. All parts of the brain and cord dorsal to it form a dorsal zone and include the receptive part of the spinal cord and medulla, as well as the cerebellum and cerebral hemispheres. This dorsal zone contains only nerve cells whose processes never leave the nerve centres to form nerves, and it receives all the entering sensory nerve fibres. It is the recipient part of the nerve centre.

The ventral zone contains all cells whose processes go out to form peripheral nerves; it has also some cells of the other type, but it does not receive any entering sensory nerve fibres. It is the efferent part of the nerve system and it includes the anterior and part of the lateral columns of the spinal cord, as well as the ventral parts of the mid-, hind-, and after-brains.*

The different segments of the brain are composed of deposits of nerve cells forming gray matter or ganglia, and of strands of nerve fibres connecting these ganglia. Most of the general description of these parts must be gotten from treatises on anatomy. I purpose, however, to enumerate and describe the various ganglionic deposits found in the brain; then show the various tracts connecting them with each other and the periphery.

* I am much indebted to Prof. C. S. Minot for details regarding this conception of the structure of the nervous system, which seems to me to deserve wider recognition.

The sixteen ganglionic deposits of the brain are arranged in the different segments as shown here:

1. Cerebral cortex
2. Corpus striatum { Caudate nucleus / Lenticular nucleus } Fore-brain. Telencephalon.
3. Olfactory lobes.
4. Optic thalamus
5. Corpora geniculata
6. Corpora mamillaria
7. Luys' body
'Tween-brain. Diencephalon.
8. Corpora quadrigemina
9. Red nucleus
10. Substantia nigra
Mid-brain. Mesencephalon.
11. Cerebellar cortex
12. Cerebellar nuclei
13. Pons nuclei
Hind-brain. Metencephalon.
14. Olivary body
15. Nodal nuclei
16. Cranial nuclei
After-brain. Myelencephalon.

The Cortex Cerebri and the Convolutions.—The gray matter of the surface of the brain is called the cortex cerebri, and it is by far the largest and most important deposit of nerve cells in the body. The cortex is from 2 to 4 mm. (one-twelfth to one-fifth of an inch) thick, and its total superficial area is 1,800 to 2,700 sq. cm. The area of gray matter lying in the fissures is about twice that lying on the surface (Donaldson). The cerebral cortex is arranged in folds or convolutions (gyri) separated by fissures or sulci. These fissures divide the brain also into lobes. The fissures of the brain are divided into *primary* and *secondary*. The former are permanent, and present little change in size, location, or direction. The latter are variable in all these respects, and are often called sulci for purposes of distinction.

The primary fissures of the brain are:

The longitudinal.
The transverse or choroidal.
The fissure of Rolando or central.
The fissure of Sylvius.
The parietal.
The parieto-occipital.
The calcarine.

The position of these fissures is indicated on the accompanying diagrams, which are based on descriptions of Eberstaller. I have not space to give detailed descriptions.

The secondary fissures or sulci will be enumerated in describing the lobes. They are divided into the typical or more or less constant and the atypical or tertiary fissures.

The primary fissures divide the cerebrum into various portions called lobes. The lobes are:

Frontal.
Parietal.
Temporal.
Occipital.
The central or island of Reil.
Olfactory.
Limbic.

There are eight fissures and seven lobes.

The frontal lobe is limited behind by the fissure of Rolando, and on the median surface by the calloso-marginal or subfrontal fissure and its ascending branch. It has the following sulci: 1. Superior frontal and superior precentral. 2. Inferior frontal and inferior precentral. 3. Olfactory. 4. Triradiate.

In the frontal lobe are the following convolutions: 1. Precentral or ascending frontal, having on its median surface (*a*) the paracentral lobule. 2. First or superior frontal. 3. Second or middle frontal. 4. Third or inferior frontal (Fig. 166).

The parietal lobe is limited behind by the parieto-occipital and

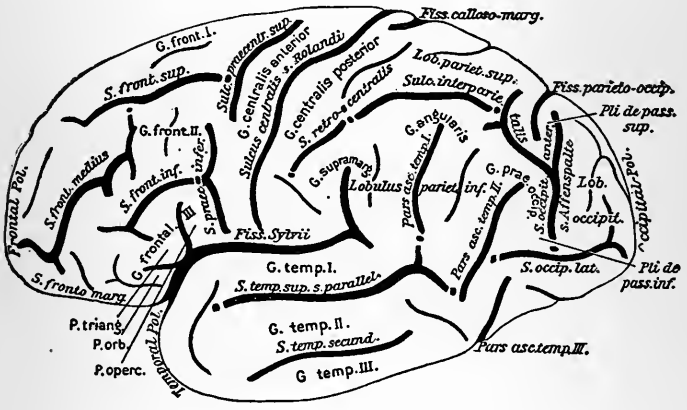


FIG. 166.—LEFT HEMISPHERE, FROM WITHOUT.

anterior occipital fissures, below on its outer surface by the Sylvian fissure and a line drawn back from its upper end to the lower part of the anterior occipital. In front it is limited by the fissure of Rolando. On its median surface it is limited in front by the ascending branch of the calloso-marginal or subfrontal fissure, behind by the parieto-occipital, and below by the subparietal fissure. Its important sulci are: 1. The parietal or interparietal. 2. The postcentral. The convolutions formed are, on the convex surface: 1. The postcentral or ascending parietal. 2. The superior parietal lobule, having on its median surface (*a*) the quadrate lobule or præcuneus. 3. The inferior parietal lobule, which is composed of (*a*) the supramarginal gyrus, (*b*) the angular gyrus. These latter gyri are variously described, but it is best to regard one, (*a*) the supramarginal, as that surrounding the posterior end of the fissure of Sylvius; the other, (*b*) the angular, as that beginning at the upper limit of the first temporal fissure and extending back as far as the anterior occipital fissure.

The Occipital Lobe.—This is limited on its convex surface, anteriorly, by the parieto-occipital fissure, laterally to this by the anterior occipital and inferior occipital fissures. These in apes form

a single continuous fissure sharply separating the occipital from the other lobes. On its median surface the parieto-occipital fissure limits the lobe in front. On the under surface the anterior edge of the tentorium about marks the anterior limit.

The sulci are: 1. The transverse occipital (ape fissure of some writers). 2. The superior or lateral occipital. 3. The inferior

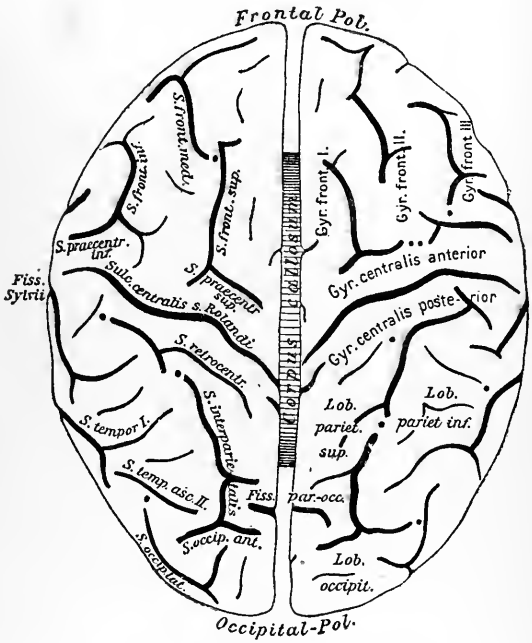


FIG. 167.—THE CEREBRUM, FROM ABOVE.

occipital. On the median surface: 4. The calcarine, which joins the parieto-occipital. 5. The inferior occipito-temporal or fourth temporal.

The convolutions on the convex surface are: 1. The superior occipital. 2. The middle occipital. 3. The inferior occipital. 4. The descending occipital. On the median surface we find: 5. The cuneus. 6. Descending occipital.

The Temporal Lobe.—The convex or lateral surface shows the following sulci: 1. The first temporal or parallel sulcus. 2. The second or middle sulcus. On the under and median surfaces are: 3. The third or inferior temporal sulcus. 4. The fourth temporal or inferior occipito-temporal or collateral sulcus, which extends into the occipital lobe. 5. The hippocampal sulcus.

The convolutions are: 1. The first temporal convolution. 2. The second temporal convolution. 3. The third temporal convolution. 4. The lateral occipito-temporal or fusiform convolution. 5.

The median occipito-temporal or lingual convolution. 6. The hippocampal convolution, which is an extension of 4 and 5. 7. The uncinate convolution, which is an extension of 6.

The Island of Reil (lobus centralis, lobus caudicis).—This has a

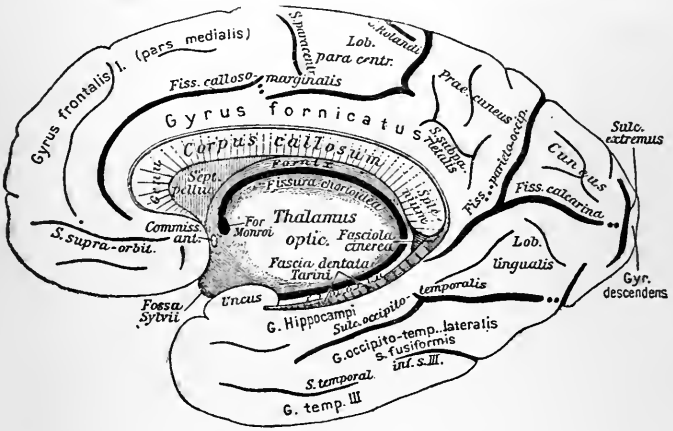


FIG. 168.—RIGHT HEMISPHERE, FROM WITHIN.

circular sulcus surrounding it, and several more or less short sulci dividing it into from five to seven short gyri.

The *limbic lobe*, if described as a separate lobe, is made to in-

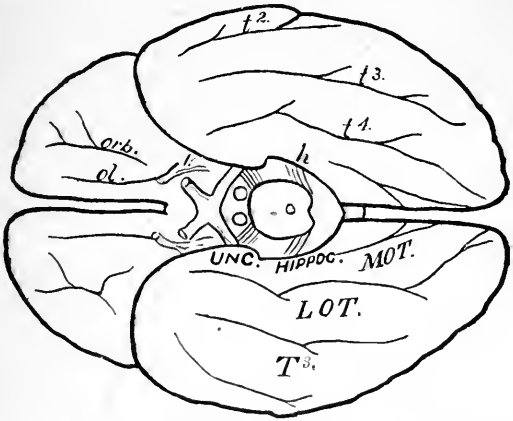


FIG. 169.—UNDER SURFACE OF BRAIN. *LOT*, lateral occipito-temporal or fusiform lobe; *MOT*, median occipito-temporal or lingual lobe.

clude anteriorly the gyrus fornicatus or gyrus cinguli; back of this the isthmus; then the hippocampal, uncinate, and dentate convolutions (Fig. 168).

The olfactory lobe is rudimentary in man. Its position is shown in the diagrams.

The operculum is the part of the brain that overlaps the island of Reil. It consists of a fronto-parietal part, formed by the lower ends of the two central convolutions, a frontal part formed by the base of the inferior frontal convolution, and a temporal part formed by the tip of the temporal lobe.

The cuneus, præcuneus, and paracentral lobule are important subdivisions on the median surface of the brain. Their position and boundaries are shown in the cuts (Fig. 168).

Microscopical Anatomy of the Convolutions.—The cortex of the cerebrum is composed of nerve cells, a network of nerve fibres and processes, and of neuroglia tissue. Superimposed upon it is a very vascular membrane, the pia mater, which sends a rich plexus of vessels into it. We shall proceed to study: 1st, the structure and arrangement of the nerve and neuroglia cells; 2d, the arrangement and connections of the nerve plexuses. On both these points new facts are being constantly added, and the present description must be in many respects only provisional.

1. *The cells* are arranged to a certain extent in layers. In the outer layer, next the pia mater, is a deposit of neuroglia tissue containing also peculiar-shaped nerve cells, called cells of Cajal. Beneath these are small, somewhat irregularly shaped pyramidal cells (angular cells of Lewis); next come large pyramidal cells; and deepest of all irregularly shaped cells (including the granule cells of Lewis) and spindle-shaped cells (Fig. 170).

In the above I have described four layers of cells, and this may be considered the type. Some anatomists describe five typical layers, the fifth being made by a subdivision of the fourth (by Meynert) or of the third (by Lewis). The common four-layer type is found in the central convolutions and frontal lobe. In the occipital region there are six (Lewis) or eight (Meynert) layers described. These are formed by the interposition of granule cells which subdivide the third layer. Various types of cortex are described, depending upon the different degree of development of the cell layers and upon the fibre arrangements. The common or motor type, as has been stated, has four layers. The large pyramidal cells are here numerous and are arranged in clusters. The sensory type has at least five layers, as seen in the occipital cortex, and here the large pyramidal cells are few and isolated.

The pyramidal cells everywhere are arranged with their apices pointing to the periphery (Fig. 170). They give off apical, lateral, and basal processes. The basal process of the pyramidal cells is continued as an axis cylinder. Some of them pass down into the white matter; others turn up and enter the fibre systems of the cortex. The spindle cells point toward the periphery also, except at the bottom of the sulci, where they lie parallel to the surface. Their processes connect neighboring areas and are called association fibres.

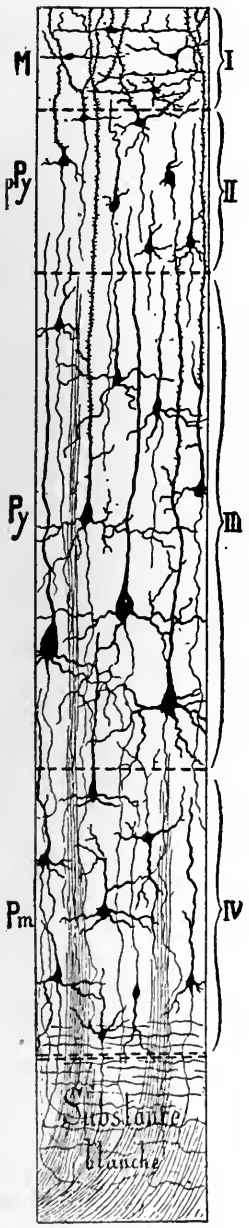


FIG. 170.

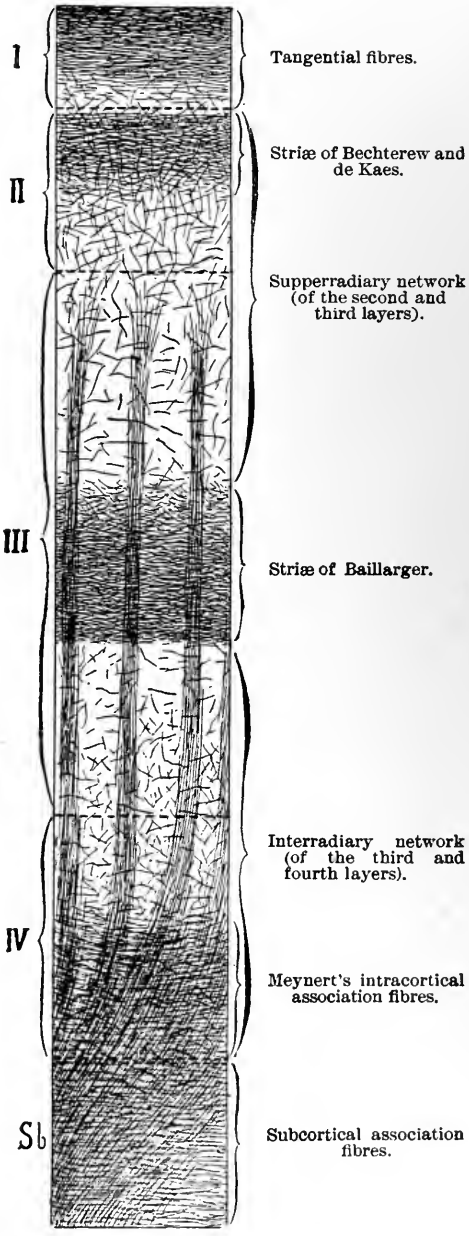


FIG. 171.

FIG. 170.—SHOWING THE DIFFERENT CELLULAR LAYERS OF THE BRAIN CORTEX (after Cajal). *M*, neuroglia layer; *pPy*, small pyramidal layer; *Py*, large pyramidal layer; *Pm*, irregularly shaped cells. The cells of Cajal can be seen in the layer *M*.

FIG. 171.—SHOWING THE LAYERS OF NERVE FIBRES. Practically the tangential fibres and the striæ of Baillarger are the only layers that show distinctly.

The small pyramidal and spindle cells measure about 10 x 18 μ; the large, 20 x 40 μ ($\frac{1}{1200}$ x $\frac{1}{600}$ in.). There are in the upper central and paracentral convolutions giant cells (of Betz) which measure 125 x 55 μ ($\frac{1}{200}$ x $\frac{1}{500}$ in.). (See Plate.)

Despite the great variety and complexity of the cortex, it is probable that there are but three principal classes of cells: (1) those which receive nervous impulses and which lie chiefly in the second and granule layers; (2) those which associate and co-ordinate these impulses, and which partly lie in the first and partly deep in the fourth layers (cells of Cajal and spindle cells); and (3) those which discharge impulses and which lie in the third layer (large pyramidal cells).

2. *The fibres and plexuses* of the cortex are composed of processes from the nerve cells and terminals coming in from the white matter. They collect into several close networks. One, lying in the neuroglia layer and running parallel to the surface, is called the *tangential* layer of fibres; a second layer runs among the large pyramidal cells, forming the outer *stripe of Baillarger;* and a third layer, beneath this, is called the inner stripe of Baillarger. Besides these, there are radiating fibres, running in from the white matter and forming interradial and superradial networks (Fig. 171).

The cortical gray matter, as will thus be seen, contains layers of nerve cells, into which nerve fibres penetrate. These terminate, as do all fibres, in end brushes, which surround the receptive or sensory cells. An enormous number of fine fibres is given off by the cells; some of these form layers in the cortex and connect neighboring parts, others run out and connect more distant parts or pass down to lower levels. There are thus three kinds of fibres—afferent, associative, and efferent—just as there are three types of cells, and since nerve cells and fibres are really parts of the same unit—the neuron—there are practically three kinds of neurons in the cortex.

The different convolutions and lobes of the brain are connected with each other by association fibres and commissural fibres and to the ganglionic masses below them by projection fibres. The association fibres consist of short fibres connecting neighboring convolutions and of long tracts which connect neighboring or distant lobes.

The *short* association fibres are numerous; they lie close beneath the gray matter and connect convolutions of the same lobes. The course of many of the *long* association paths is not yet well known. The occipital lobe is connected by long tracts to the temporal lobe, and perhaps slightly to the inferior parietal lobe (H. Sachs). The temporal lobes are connected only to the occipital. The frontal lobe is apparently connected with the parietal. Its connection with the temporal is denied (H. Sachs). All the lobes of the two halves of the brain are connected with each other by commissural tracts in the corpus callosum and anterior commissures. I will describe the *projection* fibres later.

The Corpora Striata which form the second ganglionic mass to be studied are composed of two parts: the caudate nucleus and lenticular nucleus. These two nuclei are separated dorsally by the white fibres forming the internal capsule. Below they are continuous. Their shape and relations are not easily appreciated except by actual inspection of specimens. They are indicated in the accompanying figures (172–174). The head of the caudate nucleus is connected with the gray matter of the anterior perforated space. The tail extends into the temporal lobe, where it is continuous with the gray matter of the cortex, called at this point

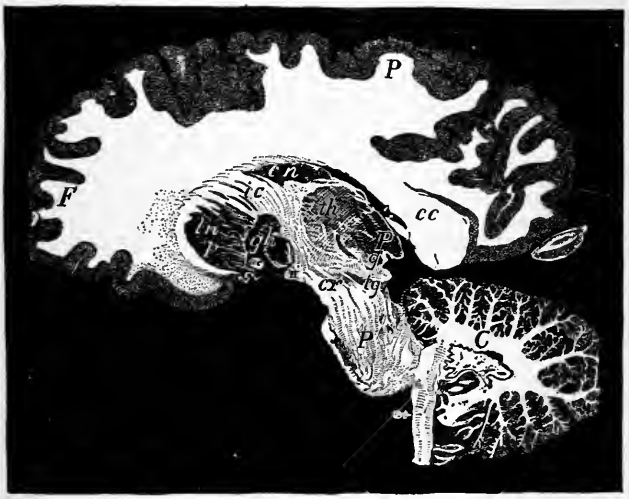


Fig. 172.—Longitudinal Section, showing the lenticular (*ln*) and caudate nuclei (*cn*) separated by the internal capsule (*ic*), the corpus callosum (*cc*), the anterior commissure (*ac*), the optic tract (*II.*), the optic thalamus (*th*), the corpora geniculata (*g*), and the pulvinar (*p*), the crusta (*cr*), the tegmentum (*tg*), pons (*P*), and cerebellum (*C*); *p*, putamen; *g*, globus pallidus (G. Stewart).

the amygdalum, and with the claustrum. The lenticular nucleus is also continuous with the gray matter of the anterior perforated space. It is divided into three portions; the external is called the putamen, the two inner the globus pallidus. These parts are separated by white fibres. The caudate nucleus, putamen, and probably all of the corpus striatum are modifications of the cerebral cortex.

The corpus striatum has (1) fibres which pass to it from the cortex, (2) fibres which pass through it from the frontal and parietal cortex, (3) fibres which originate in it connect its different parts, and go to parts below it. 1. The fibres joining the ganglion to the cortex are few and merely associative. 2. The fibres which pass through it pass chiefly into the dorsal part of the cerebral peduncles (or tegmentum) and are connected with the sensory tracts there.

3. Fibres which originate in the caudate nucleus and putamen collect together and join with the cortical fibres to form the "lenticular loop" (ansa lenticularis). Some go to the subthalamus (Luys' body) and optic thalamus; most go to the inferior olives and thence connect with the cerebellum. Some perhaps go to the posterior longitudinal bundle, which is a band of commissural fibres

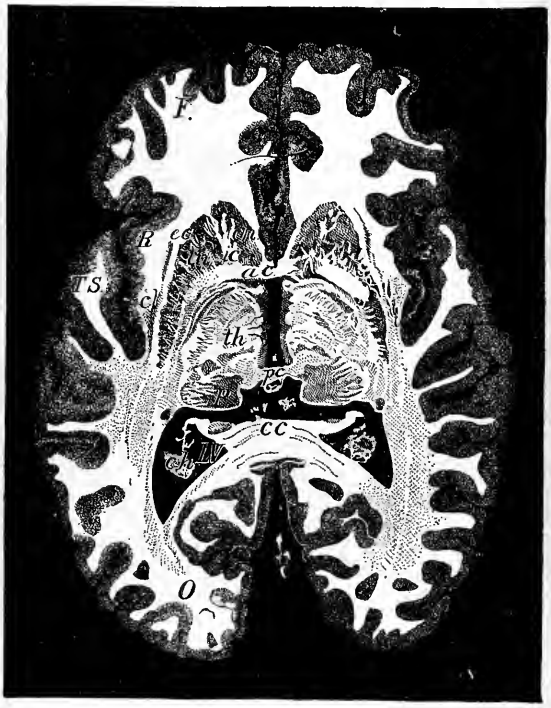


FIG. 173.—HORIZONTAL SECTION, showing the frontal (*F*), temporo-sphenoidal (*TS*), and occipital lobes (*O*), with their gray and white matter, the island of Reil (*R*), its gray and white substance, the claustrum (*cl*), the external capsule (*ec*), the lenticular nucleus (*ln*), the caudate nucleus (*cn*), the internal capsule (*ic*), the optic thalamus (*th*), the pulvinar (*p*), the corpus callosum (*cc*), the anterior and posterior commissures (*ac* and *pc*), the lateral ventricle (*LV*), with the choroid plexus (*ch*) (G. Stewart).

that connect together the cranial nerve nuclei in the medulla. Fibres connect the caudate nucleus and putamen with the globus pallidus.

The corpora striata are relatively rudimentary ganglia in man. They contain sparsely distributed multipolar and fusiform cells, $\frac{1}{1800}$ to $\frac{1}{500}$ in. in diameter, the larger being in the lenticular nucleus. They are chiefly of the second or Golgi type.

THE OPTIC THALAMI.—These ganglia form the chief part of the 'tween-brain. They lie at the base of the brain, posterior and internal to the corpora striata. They are continuous with each

other by means of the middle gray commissure. The upper or dorsal surface forms part of the wall of the lateral ventricles; the mesial surface forms the lateral wall of the third ventricle. Externally is the band of white fibres called the internal capsule; below this is a rather large mass, the red nucleus, external to a small gray nucleus called the subthalamic ganglion (Luys' body). Around and below these is a complex network of fibres called the stratum intermedium. Below this, on the base of the brain, are the corpora mamillaria (Fig. 175). The thalamus is composed of six nuclei—the anterior, lateral, median, ventral, posterior, and the *pulvinar*

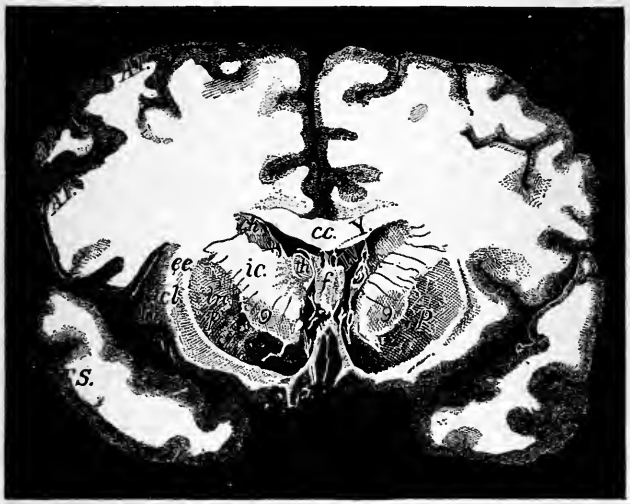


FIG. 174.—SECTION THROUGH THE MIDDLE OF THE BASAL GANGLIA, PRECENTRAL (*AF*), AND PART OF POSTCENTRAL (*AP*) CONVOLUTIONS, AND (*TS*) TEMPORAL LOBE. It shows the corpus callosum (*cc*), the fornix (*b*), the fifth ventricle (*Y*), lateral ventricles (*LV*), small part of thalamus (*th*), internal capsule (*ic*), caudate nucleus (*ch*), lenticular nucleus (*ln*), external capsule (*ee*), claustrum (*cl*) (G. Stewart).

(Fig. 172, p. 353). Besides these there is on the median surface a small ganglion called the g. habenulæ. The optic tracts wind around the posterior and outer edge of the thalamus; and connected with these and the thalamus are two other ganglia, the external or lateral and internal or median *geniculate bodies*. Thus the thalamus is in close anatomical relations with five small ganglia; three below it—the corpus mamillare, red nucleus, and subthalamus; two postero-external to it, the geniculate bodies. To these may be added the corpora quadrigemina. The thalamus is composed of multipolar nerve cells rather more numerous and larger than those in the striatum, but not grouped closely together. Starr describes a small stellate multipolar cell and a large multipolar cell. There are, besides these, cells of the second or Golgi type which have associative functions. The optic thalamus is composed of

nerve cells whose neuraxons pass up to the cortex; and of the terminal fibres from neurons lying in the cortex and in the sensory cranial and spinal nuclei below. Between these are the Golgi or

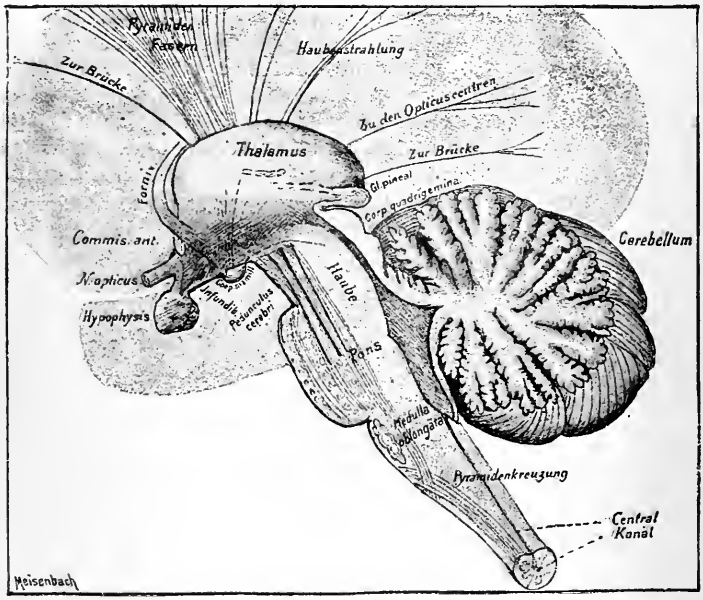


FIG. 175.—SHOWING THE RELATIONS OF THE OPTIC THALAMUS TO OTHER PARTS (Edinger).

associative cells which unite these various afferent and efferent neurons. The optic thalamus is a terminal station of numerous fibres of sensation coming from the medulla and cord; it is also

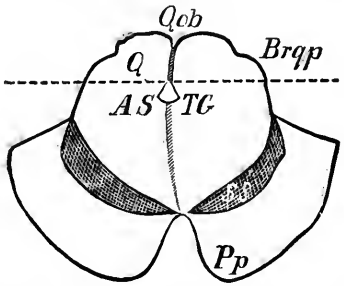


FIG. 176.—SECTION THROUGH THE CORPORA QUADRIGEMINA (*Q*), TEGMENTUM (*TG*), AND CEREBRAL PEDUNCLES. *SS*, Substantia nigra; *Pp*, peduncles; *AS*, aqueduct of Sylvius.

the place of origin of cells which send processes to the cortex and the receiving centre of fibres coming from the cortex. It is therefore a very important sensory-motor ganglion connected with

the reception and distribution of sensory and motor influence and with the automatic and reflex movements.

The Corpora Quadrigemina.— The mid-brain contains as its chief ganglia the corpora quadrigemina. These consist of four tubercles, two anterior and two posterior (Fig. 175). In man they are rudimentary in structure and relatively unimportant in function. In man also the posterior tubercles are developed more relatively than in most lower animals. They together measure about 14 mm. ($\frac{1}{2}$ in.) in sagittal direction. In front lie the pineal gland and

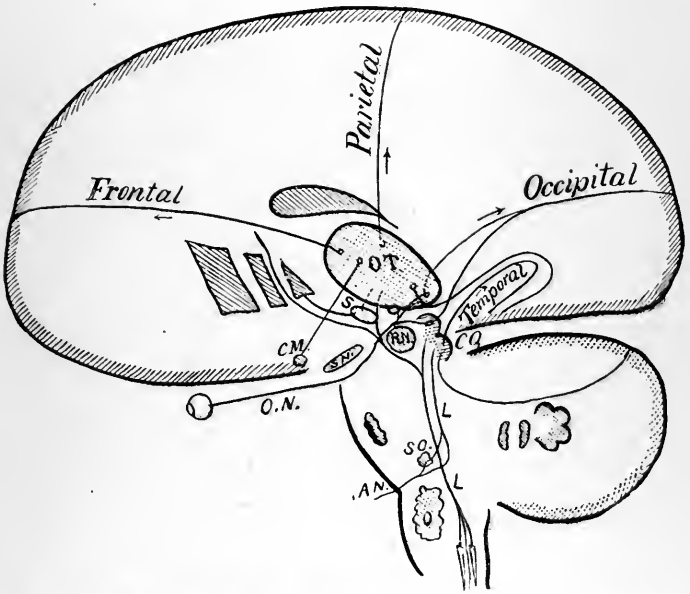


Fig. 177.—Showing Some of the Relations of the Corpora Quadrigemina and Optic Thalamus to Other Parts. *OT*, optic thalamus; *CQ*, corp. quad.; *RN*, red nucleus; *S*, Luys' body; *SN*, substantia nigra; *L*, sensory fibres from cord; *CM*, corp. mamillare.

third ventricle; beneath are the aqueduct of Sylvius and the structures of the upper pons and cerebral peduncles. This inferior boundary is shown by the dotted line in the cut (Fig. 176). Microscopically the anterior lobes consist of layers suggesting a cortical type (Spitzka); the outermost is made up of optic-tract fibres, the next is a thin layer of small nerve cells, then optic fibres again, and deepest of all a layer containing a few large cells. The posterior lobes are more homogeneous and contain small multipolar cells and a ganglion.

The figures 172 to 174 show the relationships of the basal ganglia and other parts.

The Substantia Nigra.—Below the corpora quadrigemina and lying between the upper sensory part (*tegmentum*) of the peduncles

of the brain and the lower motor part (*crusta*) is the substantia nigra. It contains large multipolar, angular, and fusiform cells deeply pigmented (Fig. 176).

The Red Nucleus.—Dorsal to the s. nigra and at about its middle extent is the red nucleus. It is spherical or oblong, very vascular, and contains numerous small cells.

The subthalamus (Luys' body) lies more dorsally but in about the same plane as the substantia nigra. It measures about $\frac{1}{3}$ by $\frac{1}{2}$ inch, and contains a few cells and a very fine plexus of nerve fibres.

The position and relation of these bodies are shown in Fig. 174.

The nuclei of the pons Varolii are irregularly distributed masses of nerve cells lying deep among the longitudinal and transverse

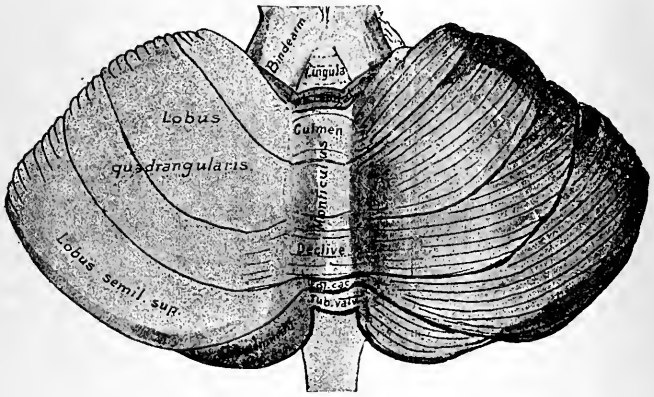


FIG. 178.—SHOWING THE LOBES AND OTHER SUBDIVISIONS OF THE CEREBELLUM, DORSAL SURFACE (Edinger).

fibres. A special nucleus lying low down in the pons is known as the *superior olive.* In the *after-brain or medulla* we have the gray matter of the floor of the fourth ventricle and its cranial nerve nuclei, the olivary bodies, and certain small deposits of gray matter called the *nodal nuclei.*

THE CEREBELLUM in man consists of a median portion or vermis and two lateral hemispheres. It is connected to the cord and rest of the brain by anterior, middle, and posterior peduncles. It is composed of an external layer or cortex of gray matter, of central white matter, and central ganglia. The gray matter lies in very close, narrow folds, producing with the white matter an appearance on section called the *arbor vitæ.* The vermis and hemispheres are divided by sulci into a number of lobes and lobules. The vermis is divided into superior and inferior portions. Its further subdivisions and those of the hemispheres are shown in the cuts (Figs. 178, 179). In the white matter of either hemisphere is a nucleus of small multipolar cells, the corpus dentatus or ciliary body. To the median side of this, and belonging structurally to it, is a small nucleus, the

emboliform nucleus. In the inferior vermis is a collection of larger multipolar cells, the *nucleus fastigium* or tegmental nucleus; just to the outer side, between it and the emboliform nucleus, is a small collection of cells, resembling those of the *n. fastigii*, called the nucleus globosus.

The Cerebellar Cortex.—If a section be taken through the gray matter of the cerebellum, it will be found to be composed of two

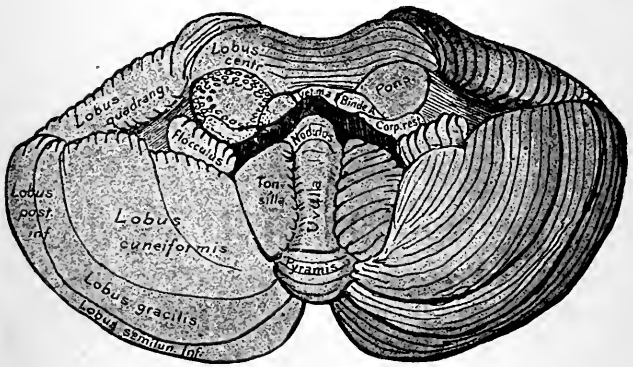


FIG. 179.—CEREBELLUM, VENTRAL SURFACE (Edinger).

layers, an outer, or molecular, and an inner, or granular, layer. Each of these layers contains a large number of peculiar-shaped nerve cells, and very rich plexus of nerve fibres. The molecular

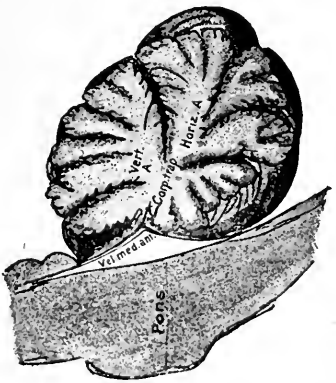


FIG. 180.—SECTION THROUGH MIDDLE OF VERMIS AND PONS.

layer contains two kinds of cells, one large and known as *Purkinje's* cells, the other smaller and known as *stellate* cells. The cells of Purkinje lie the more deeply, being, in fact, practically at the boundary of the molecular and granular layers. They measure 40 x 30 μ ($\frac{1}{6\,0\,0}$ to $\frac{1}{8\,0\,0}$ in.) and have large round nuclei. Each cell gives

off an enormous number of branching dendrites, which run up toward the surface of the cerebellum in the shape of a bush. Each little branch sends off from the side small buds, which are called the *gemmules* or thorns. These branching dendrites do not pass up altogether like the branches of a round bush, but are flattened like a broom. The Purkinje cells give off from their base a neuraxon which runs down into the white matter of the cerebellum (Fig. 181, *P*).

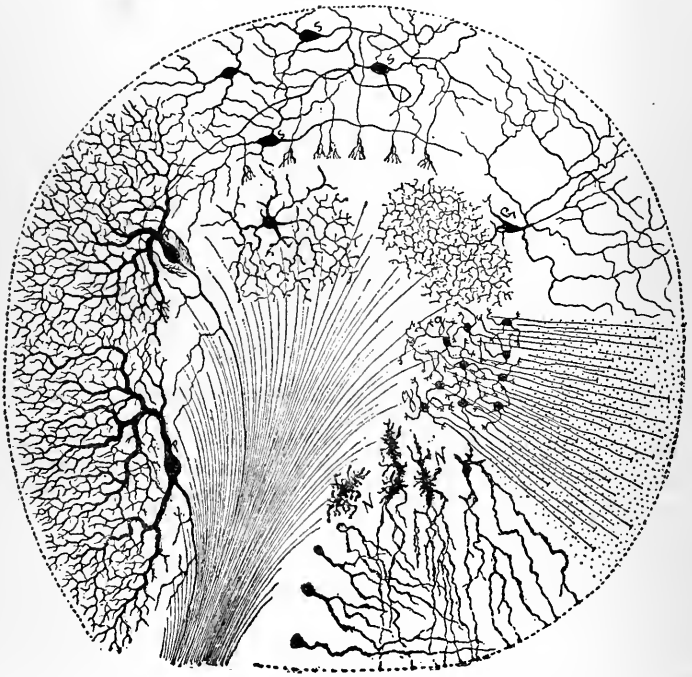


FIG. 181.—THE DIFFERENT CONSTITUENT ELEMENTS OF THE GRAY CORTICAL LAYER OF THE CEREBELLUM.

Lying throughout the molecular layer are the *stellate cells*, which are much smaller in size, and which also give off a number of dendrites (Fig. 181, *S*). Each cell has also its axis cylinder (neuraxon) and this sends off collaterals which end in a fine basket-like network which surrounds the body of the cells of Purkinje (Fig. 181). On this account they are sometimes called basket cells. There are other stellate-shaped cells in the molecular layer which lie more superficially, and do not have this particular connection with the Purkinje cells, but appear to belong to the same type.

The granular layer contains a large number of very small granular-like cells that Golgi was the first to show were really nerve cells. They are only about 5 μ ($\frac{1}{5000}$ in.) in diameter, and they have a number of short dendrites which end in clubbed extremities. (Fig. 181, *G*). They give off a very fine axis-cylinder process (neu-

raxon) which runs up into the molecular layer and there divides in a T-shaped fashion, the fibres running parallel to the surface of the convolution and passing in between the branches of the cells of Purkinje. There are besides these granular cells a few larger cells with axis cylinders that divide and subdivide, ending in a finely ramifying plexus. These are of the type known as the *cells of Golgi*. They are found in other parts of the brain.

The nerve fibres of the cerebellar cortex are, as in the cerebrum, radiating and tangential. The tangential fibres lie at the level of the Purinkje cells, among the processes of which they run. Just

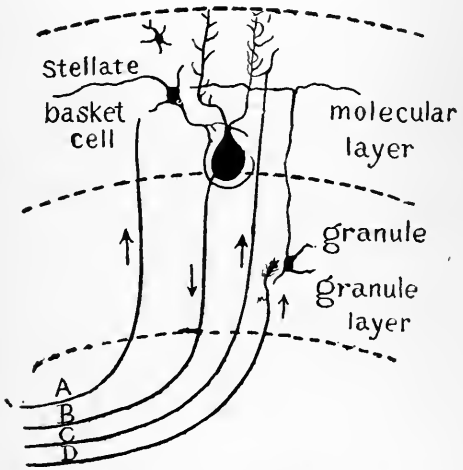


FIG. 182.—SCHEMATIC DIAGRAM OF THE RELATIONS OF THE CEREBELLAR CELLS. *A*, Afferent fibre to basket (stellate) cell; *B*, neuraxon of Purkinje cell; *C*, afferent fibre to Purkinje cell; *D*, afferent (mossy) fibre to granule cell.

beneath the pia is a thin connective or neuroglia tissue membrane which sends radial fibres down through the gray matter, affording it a support.

It will be seen that the general arrangement of the cerebellar cortex is analogous to that of the cerebrum. Associative and receptive cells are found in the granular and molecular layers, and they send processes forming a rich network around Purkinje's cells, which are efferent in function (Fig. 182). The comparatively small number of the large cells is in harmony with the view that the cerebellum is an organ that receives and adjusts nerve impulses for co-ordinate distribution. All parts of the cerebellar cortex are anatomically alike.

The white matter of the cerebellum consists of nerve fibres, some of which go to form the peduncles. Others form anterior and posterior commissures, running through the two extremities of the vermis and connecting the hemispheres. There is also a longitudinal commissure in the vermis. The white matter around the corpus dentatum is called the *fleece*.

The Projection System.

Having described the general arrangement of the different divisions of the brain and the collections of gray matter found in them, we are prepared to study the tracts of white matter which connect the different parts.

The white matter, as already shown, is made up of:

1. Association fibres { Short. Long. Commissural.
2. Projection fibres.

1. The association fibres of the cortex have been already described.

2. The *projection fibres* are those which connect different areas of the cortex with the basal ganglia and the ganglionic masses of the pons, medulla, and spinal cord. Recent researches by Flechsig have led him to assert that the projection fibres are much less numerous than has been suspected, and that they come only from the central convolutions, part of the first and second temporal, part of the occipital lobe, the hippocampus, uncus, and part of the limbic lobe. These are the parts of the brain identified with the function of voluntary movement and general and tactile sensation, hearing, sight, smell, and taste. The area of the cortex of the brain thus connected to parts below by projection fibres is only about one-third of the whole. The remaining two-thirds of the brain cortex is not in direct connection with parts below, but is closely connected with the projection centres by association fibres. Flechsig considers the parts of the brain which are thus connected by association fibres as higher centres, identical with the more complex mental acts, and he calls them the *association centres*.

The association centres according to this view are the frontal lobes, part of the parietal and occipital and part of the temporal lobes. The views of Flechsig have attracted great attention, but have not yet been generally adopted, and may require considerable modification.

The projection fibres of the brain form the different pathways (a) by which the special and general sensations pass up to the brain cortex and (b) by which the voluntary, automatic, and psycho-reflex movements of the body are brought about.

(*b*) *The Motor Tracts.*—The cerebro-spinal motor paths are of two kinds: the direct or voluntary and the indirect motor tracts. The *direct motor tract* originates from cells in the central convolutions; the neuraxons of these cells pass down and are gathered together in a narrow band which passes through and occupies nearly the whole of the posterior segment of the internal capsule. The fibres continue on into the pons Varolii and medulla, and at the latter point give off some terminals which cross (except those for the sixth nerve) to the nuclei of the motor cranial nerves. The rest of

the bundle passes on through the medulla, and ninety per cent cross over at its lower portion, forming there the anterior pyramids. About ten per cent of the fibres do not cross, however, but continue on the same side. The crossed bundle passes into the lateral column of the spinal cord, forming the crossed pyramidal tract, which passes on, diminishing in size as far as the sacral part of the cord. It gives off terminal end brushes which surround the

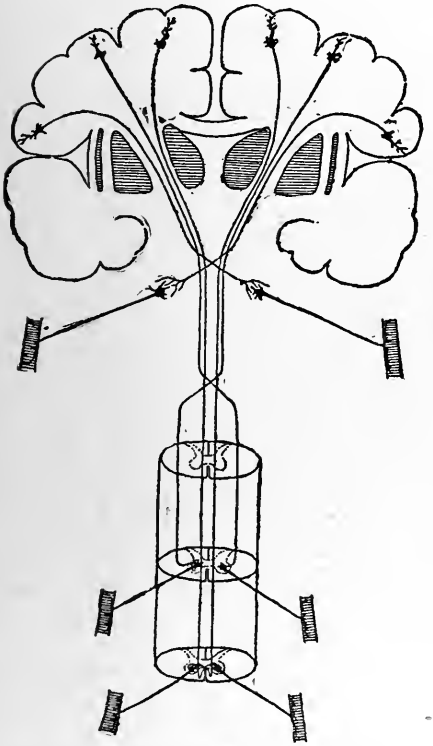


FIG. 183.—DIAGRAM OF THE DIRECT OR VOLUNTARY MOTOR TRACT, showing the course of the motor impulses from the cerebral cortex to the voluntary muscles (after Van Gehuchten).

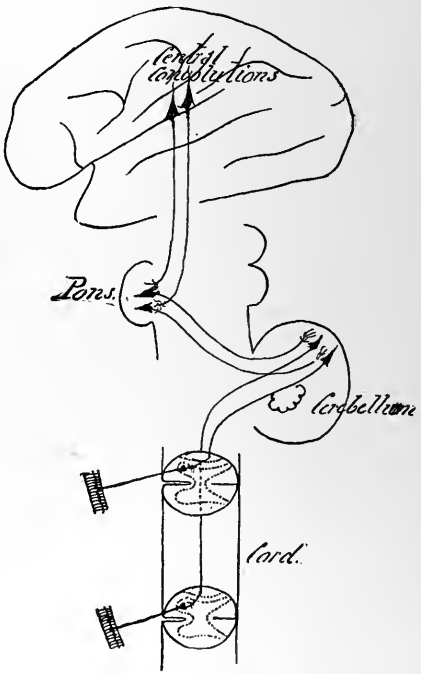


FIG. 184.—DIAGRAM OF THE INDIRECT OR INVOLUNTARY MOTOR TRACT.

cells of the anterior horns. The small uncrossed band of fibres continues on in the mesial part of the anterior column, forming the direct pyramidal tract, or column of Türck. The fibres of this tract cross over in the anterior commissure at different levels, and their terminals also connect with the motor cells of the anterior horns. Thus it will be seen that the direct motor tract is a long continuous strand of fibres, composed of single neurons putting the cortex of the central convolution directly in contact with the motor cells of the pons, medulla, and spinal cord of the opposite side.

Each pyramidal tract, as it reaches the spinal cord, contains about eighty thousand fibres (Fig. 183).

The indirect motor tract arises from nerve cells in the anterior central convolution, and, perhaps to some extent in the frontal lobe adjoining. Its fibres pass down into the internal capsule, mingling directly with those of the direct motor tract and giving off collaterals to the optic thalamus. The fibres pass through the cerebral peduncles, occupying their inner four-fifths, or motor part, and, finally, reach certain deposits of nerve cells in the pons Varolii known as the pons nuclei. They surround these cells here with terminal end brushes. From these cells neuraxons cross the median line in the middle cerebellar peduncle and thence to the cortex of the cerebellum, where they in turn end. From here the nerve cells send fibres through the peduncles by paths not perfectly well known down into the spinal cord, where they pass along mainly in the lateral fundamental columns, to connect finally with the anterior-horn cells. Thus it will be seen that the indirect motor tract is composed of (*a*) a cortico-pontine neuron, (*b*) a pons-cerebellar neuron, (*c*) a cerebello-spinal neuron, and (*d*) the peripheral motor neuron (Fig. 184).

The direct motor tract is concerned in all voluntary movements, and when the anterior-horn cells of the cord are cut off from it by disease there is a spastic form of paralysis. The indirect motor tract is concerned in the co-ordination of bodily movement and in the higher reflex and automatic acts. It is largely through these indirect tracts that the skilled automatic movements take place. Playing musical instruments and the involuntary use of the hands and limbs in work or games of skill are under the control of this mechanism. When the spinal cord is cut off from it, there is an unsteady and disordered gait and arm movement. The course of the direct and indirect motor tracts is shown in the accompanying diagrams.

The Sensory Tracts.—The next important pathways in the brain and cord are the sensory, and they are concerned in bringing tactile, muscular and general sensations from the remoter parts of the body to the cortex of the brain. It will be easier to follow these tracts if we begin at the periphery and follow the course of the fibres up to their centres in the brain. Just as in the case of motor fibres, we find direct and indirect tracts, although here even the direct sensory path is more tortuous and broken than is the case with the motor tracts.

The Direct Sensory Tract.—A tactile irritation of the skin passes up the sensory nerve to a posterior spinal ganglion, where the cell body from which the fibre is derived is found. It passes directly through the ganglion, enters the posterior root of the spinal cord, and passes up to a group of cells lying in the posterior horns, where it meets and surrounds with its end brush a second sensory cell. A second neuraxon starts from the body of this cell; it crosses over through the anterior commissure of the spinal cord to the

lateral column of the other side, where it runs up in the antero-lateral ascending tract, reaches the medulla and pons, and passes through this until it comes to the optic thalamus. Here it sends its terminal to a third cell, which in turn sends a neuraxon to the cortex of the central convolutions. Thus the cortical centres of the direct

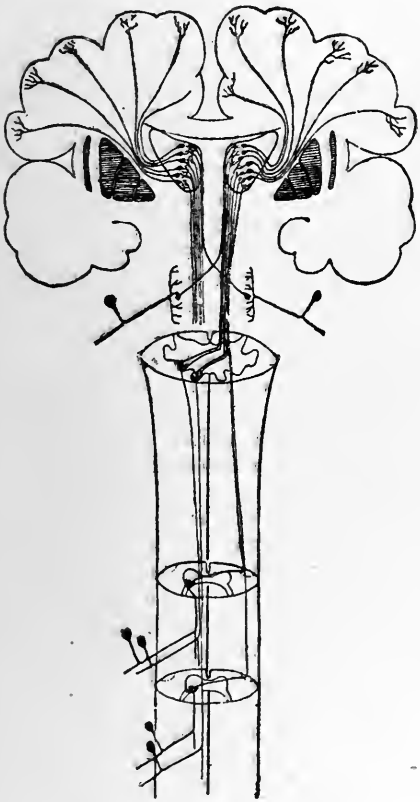


FIG. 185.—THE DIRECT SENSORY TRACT, showing the arrangements of the neurons (Van Gehuchten).

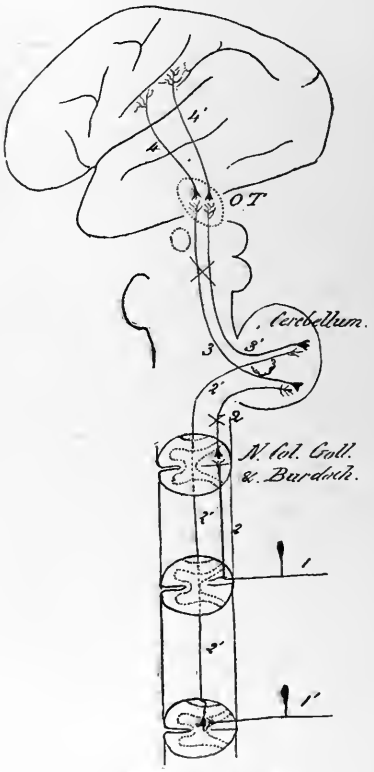


FIG. 186.—THE INDIRECT SENSORY TRACT.

sensory path are practically the same as the motor area from which the motor tract started. The direct sensory tract is made up of (1) a peripheral sensory neuron, (2) a spinal-thalamic neuron, (3) a thalamic-cortical neuraxon (see Fig. 185).*

The indirect sensory tract conveys impulses which originate in muscles, joints, and the viscera. The impulses pass up sensory

* There are also some direct sensory impulses which go from the posterior roots to the columns of Goll and Burdach and thence to the optic thalami and central convolutions.

nerves through the posterior roots. Some now pass directly into the posterior column of the cord of the same side and ascend till they reach the upper end, where their end brushes surround the cells of the nuclei of the column of Goll and of Burdach. From there they cross over to the other side in the sensory decussation. Some then go to the cortex of the cerebellum, where they terminate. The cerebellar cells take up the impulse and transmit it through the superior cerebellar peduncles to the red nuclei and optic thalamus, where they terminate. Another neuron now carries the impulse on to the central convolutions. Other indirect sensory impulses go from the sensory roots to the cells of the column of Clark, thence by the direct cerebellar tracts to the cerebellum, thence to the red nuclei and thalamus, and finally to the brain cortex. The indirect sensory tract is thus composed of (1) a peripheral sensory neuron, (2) a spinal-cerebellar neuron, (3) a cerebellar-thalamic, and (4) a thalamic-cortex neuron. The direct sensory tracts carry, for the most part, the sense of touch, pain, and temperature. The indirect sensory tracts are concerned with the sensation from the muscles and joints which have to do with co-ordination, and also with visceral sensations. It is through the indirect sensory and indirect motor tracts that the automatic and psycho-reflex acts are performed.

Other Projection Systems.—The optic, acoustic, and olfactory projection tracts have been described in connection with their peripheral nerves.

The Membranes of the Brain.—The membranes of the brain are the dura mater, the arachnoid, and pia mater. The dura mater lines the inner surface of the skull. It is attached loosely to the concavity, but closely to the base. It splits into two layers to form the venous sinuses of the skull. The inner of the two layers at certain points projects inward to form membranous septa. These are known as the great longitudinal or cerebral falx, the lesser longitudinal or cerebellar falx, and the tentorium. Hence both venous sinuses and membranous septa are formed out of the inner layer. The outer layer forms the periosteum of the bone. The dura mater is supplied with sensory nerves, chiefly by the trigeminus but posteriorly by the vagus. The blood supply will be described later.

The arachnoid is a thin, transparent, fibrous, non-vascular membrane lying between the pia and dura and continuous with the spinal arachnoid. It bridges over the fissures and the depressions at the base of the brain and forms between the pia and itself certain lacunæ or spaces. These are the central lacuna found at the beginning of the fissure of Sylvius, the callosal, and those of the transverse fissures and of the lateral aspect of the pons Varolii. The space between the dura and arachnoid is called the subdural or arachnoid cavity. It is lined with epithelium and resembles other serous cavities. The inner surface of the arachnoid is connected with the pia by numerous delicate fibrous processes. The space between these membranes is called the *subarachnoid space*. It com-

municates with the subdural space by means of the foramen of Magendie, which lies in the part of the arachnoid that passes over the pons and medulla, closing in the fourth ventricle. The subdural and subarachnoid spaces contain a serous fluid. The normal amount ranges from two drachms to two ounces, it being greater in old people. The arachnoid contains no nerves or blood-vessels. It is described by some as a part of the pia mater.

The pia mater lies beneath the arachnoid and is closely applied to the brain in all its folds. It is continuous with the spinal pia. It is very vascular and supplies the whole periphery and part of the interior of the brain with blood. It consists of two layers: an outer holding the larger vessels, and an inner delicate layer closely associated with the superficial neuroglia of the brain. The pia mater folds upon itself and passes through the transverse fissure into the third and lateral ventricles of the brain. These vascular folds form the velum interpositum, which gives off a choroid plexus to the lateral and third ventricles. Another fold, the inferior choroid plexus, is given off to the fourth ventricle. The pia mater has vasomotor, but no sensory nerves.

Functions of the Brain Membrane.—The dura mater, by its outer layer, acts as a periosteum; by its inner layer as a lymph sac. It is also, by virtue of its sensitiveness, a protection against injury and disease. The arachnoid forms the inner wall of the lymph sac. The pia mater is a vascular and nutritive organ. It is, however, also closely connected with the lymphatic system of the arachnoid. The blood supply and lymph supply of the brain vary in amount. In congestion the lymph can pass into the spinal canal or be rapidly taken up by the absorbents. In anæmia there may be compensatory increase of lymph. This fluid in disease may accumulate in the arachnoid sac, the subarachnoid space, or the ventricles, these spaces being all in communication with each other.

THE BLOOD SUPPLY OF THE BRAIN AND ITS MEMBRANES.—The vascular supply of the scalp, skull, and dura mater comes from the external carotids; that of the eye, brain, and pia mater from the internal carotids and vertebrals. The arrangement is shown here:

- External carotid gives off
  - Occipital, inferior meningeal, arteries.
  - Posterior auricular.
  - Temporal
    - Anterior.
    - Middle.
    - Posterior.
  - Ascending pharyngeal, posterior meningeal.
  - Internal maxillary, middle meningeal, small meningeal.
- Internal carotid gives off
  - Anterior meningeal.
  - Anterior cerebral.
  - Middle cerebral.
  - Posterior communicating.
  - Anterior choroid.
- Vertebral and basilar give off
  - Posterior meningeal.
  - Inferior cerebellar.
  - Anterior cerebellar.
  - Superior cerebellar.
  - Posterior cerebral.

The general arrangement and distribution of the arteries of the scalp and dura are shown in the accompanying diagram (Fig. 187).

The *blood supply of the meninges* comes from the anterior, middle, and posterior meningeal arteries. These all come, except the small anterior meningeal branches and a small posterior branch, from the external carotid. The blood passes into the diploic veins, and from there passes chiefly into the lower occipital and lateral sinuses.

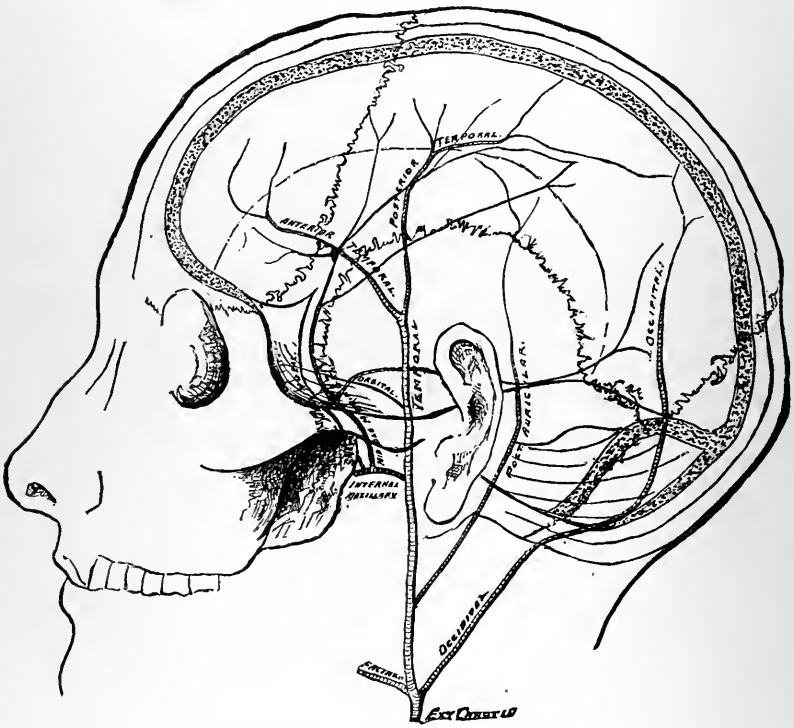


FIG. 187.—SHOWING THE BLOOD SUPPLY OF THE SCALP AND THAT OF THE DURA MATER BY THE MIDDLE MENINGEAL.

Some of it, however, returns in the venæ comites. It all returns down toward the base of the skull. The most important of the arteries is the middle meningeal, both on account of its size and its distribution above important functional areas.

The *blood supply to the pia mater and brain substance* comes from the internal carotid and the vertebral arteries. The branches of the former artery give off the anterior and middle cerebral, the posterior communicating, and anterior choroid. The vertebral arteries give off the inferior cerebellar, while the basilar branch of the vertebrals gives off the transverse, anterior cerebellar, superior cerebellar, and posterior cerebral arteries.

The cerebral arteries, anterior, middle, and posterior, are the three largest and most important. By their anastomoses the circle of Willis is formed (Fig. 188). From the circle of Willis and the beginnings of the three arteries mentioned, several groups of vessels, six in all, are given off. They enter the base of the brain and supply

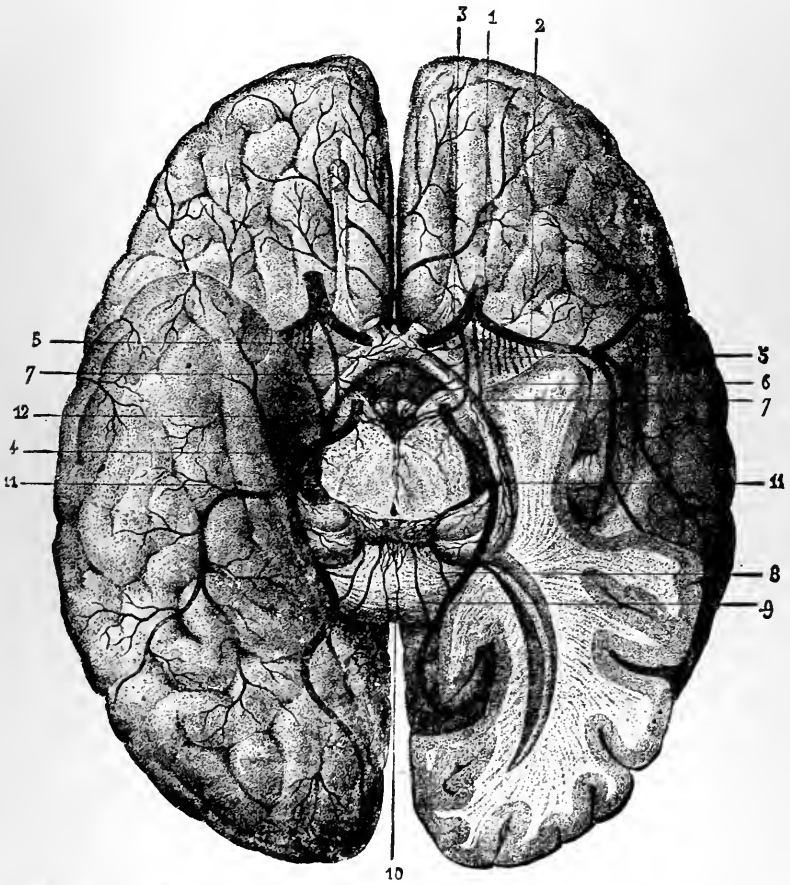


FIG. 188.—SHOWING THE ARTERIES AT THE BASE OF THE BRAIN. On the right side the brain is cut away, showing the cerebral arteries and the course of the posterior cerebral.

the great basal ganglia and adjacent white matter. They are called the *central arteries*, and they are the vessels usually affected in cerebral hemorrhages of adult life. They do not anastomose with each other. The *cortical arteries* are the terminal branches of the great cerebral arteries. They anastomose with each other but slightly. They are distributed very widely and carry much more blood than the central groups. Their distribution is shown in Fig. 189. The cortical arteries are distributed in the pia, and from there they pass

in two sets, a superficial and a deep, into the gray matter, and for a short distance into the white matter. They pass straight in at right angles to the surface. They have richly arborescent branches which do not anastomose; consequently a knife plunged straight into the brain does not cut many vessels. The cortical arteries probably anastomose somewhat with each other, though not very freely. There is slight if any anastomosis between the cortical and central arteries. The pressure is thought to be less in the vessels of the gray matter.

The capillaries are surrounded by spaces called perivascular spaces which serve as lymphatic channels. The neuroglia cells send

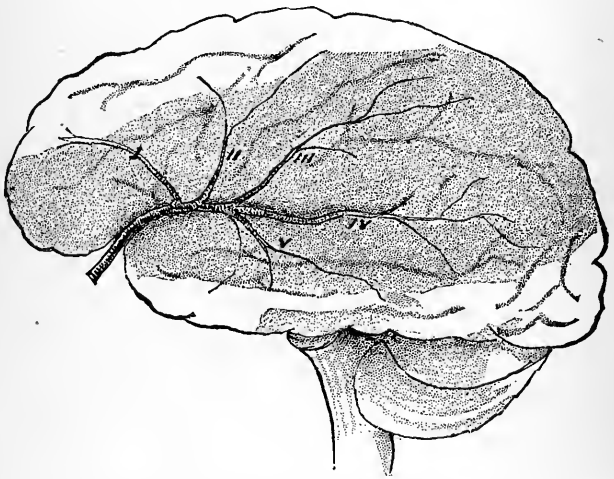


FIG. 189.—SHOWING THE DISTRIBUTION OF THE ARTERY OF THE SYLVIAN FISSURE, A PROLONGATION OF THE MIDDLE CEREBRAL. The area in front of the shaded part is supplied by the anterior cerebral, that behind by the posterior cerebral.

processes which connect with or form passages to the vessel walls (Fig. 190). The blood-vessels of the brain have probably vasomotor nerves, though this is denied by some.

The blood of the convex and mesial cerebral surface, flowing up from the base, leaves the capillaries and enters veins. Thence it still passes upward, and for the most part enters the longitudinal sinus. The most of the vessels enter the posterior portion of the sinus and in a direction forward and upward, *i.e.*, against the current in the sinus. The course of the blood current is, therefore, opposed both to gravitation and to the venous flow.

The *veins of the cerebrum* are: 1, the superficial cerebral; 2, the deep cerebral veins; and, 3, the cerebral sinuses. The superficial cerebral veins are venæ comites. Those on the convex and mesial surfaces empty chiefly into the superior longitudinal sinus, as described; those on the basal surface empty into the cavernous and lateral sinuses. These veins have no valves, and their walls are

very thin and without muscular fibres. The deep cerebral veins, or venæ Galeni, receive the blood from the lateral ventricles and from some of the central arteries supplying the basal ganglia. They empty into the straight sinus.

The *cerebral sinuses* are fifteen in number. The important ones are the superior and inferior longitudinal, the straight, the lateral, the occipital, the cavernous, and the superior and inferior petrosal. They carry blood for the most part in a direction from before backward, and convey it eventually to the internal jugular.

Most of the blood of the convexity and mesial surface must pass into the longitudinal sinus, but there is a slight connection of some

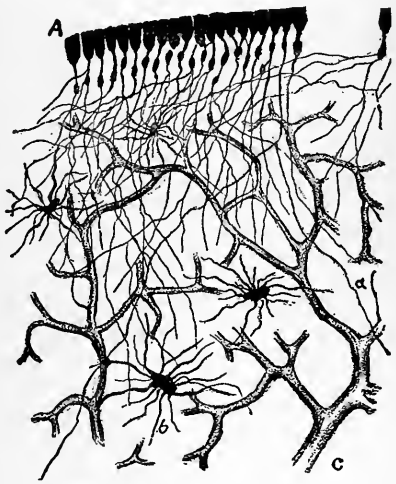


FIG. 190.—SHOWING THE NEUROGLIA CELLS OF THE BRAIN, THEIR RELATIONS TO THE BLOOD-VESSELS; ALSO THE SUSTENTACULAR PROCESSES OF THE EPITHELIAL CELLS OF THE LATERAL VENTRICLE (Marchi). *A*, Epithelial cells lining lateral ventricle; *a*, process of same; *b*, spider or neuroglia cell; *c*, blood-vessel.

of the veins with the superior petrosal and straight sinuses. The superior longitudinal sinus also communicates slightly with veins of the scalp and with the facial vein. Some of the blood from the mesial surface also goes to the veins of Galen.

On the whole, however, the system of the convex and mesial cerebral surface is a close corporation, the blood having to pass into the superior longitudinal sinus and torcular Herophili, where it meets that of the straight and occipital sinuses, and flows forward through the lateral sinuses to the internal jugular. The circulation of the basal surface is less isolated. All the basal sinuses communicate with each other freely, and there are slight communications between the veins of the scalp and the cavernous, lateral, and inferior petrosal sinuses. It is safe to tie any of the sinuses, except the lateral and the posterior part of the longitudinal. The cerebellar veins, superior, inferior, and lateral, empty into the straight,

the lateral, and superior pertrosal sinuses. None of the cerebral veins or sinuses have valves.

The pressure in the internal carotid arteries is about 150 mm., that in the cerebral sinuses 70 to 80 mm. (Gerhardt), and that in the jugular veins is almost negative. Both arteries and veins are more delicate than the extracerebral vessels.

Except in gray matter, the brain is not a very vascular organ, but this gray tissue ranks in richness of blood supply with the lungs and liver. The amount of blood in the brain at any one time is only about one to two per cent of the total blood in the circulation, or about four ounces (Ranke).

The diameter of the common carotids is 6.7 mm. (Thorne), that of the subclavians 6.2 mm., that of the internal carotids 4 mm., and that of the vertebrals 3.55 mm. (Gerhardt).*

The Functions of the Brain—Cerebral Localization.—The brain is the seat of conscious intelligence and mental activity. It has also control and direction of voluntary movements, it is the seat of instinctive acts, and it regulates in a measure the vasomotor, trophic, and secretory mechanisms of the body.

The Prefrontal Lobes.—The prefrontal lobes, or that part of the brain in front of the precentral convolution, are concerned with volition and the power of self-control, concentration of thought and attention (Ferrier). They form one of the higher or association centres. The posterior part contains centres for the movements of the head and eyes. Injuries in this part of the brain produce changes of character, indicated by peevishness and irritability of temper, mental enfeeblement, lack of power to concentrate the mind or to control the acts or emotions.

The Central Convolutions.—This part of the brain is called the *sensori-motor area,* because it is concerned in the production of nervous impulses which cause voluntary motions of the body. Certain parts of this area are in relation with certain groups of voluntary muscles on the opposite side of the body. These areas preside not so much over single muscles as over those groups of muscles which act together in producing definite purposeful acts. The lower part of the central convolutions, known as the central operculum, is a centre for movements of the larynx, mouth, tongue, and face. Above this area and about the middle third of the central convolutions is the centre for the movements of the shoulder, arm, hand, and fingers. Still farther up, near the longitudinal fissure, and extending over into the mesial surface and back into the superior parietal lobule, is the area for the trunk, hips, legs, feet, and toes. The base of the

* J. Crichton Brown gives the last two diameters 2.8 and 2.2 mm. respectively.

first and second frontal convolutions is the centre for movements of the head and eyes. The exact arrangement of these centres, which have been determined by experiments upon monkeys and other lower animals as well as by clinical and surgical observations on man, is shown in the accompanying Figs. 191, 192. The motor area is also the centre for the cutaneous sensations of the parts corresponding to the muscular groups which it supplies, so that what is called the motor is really a sensori-motor area. The motor area, when irritated by disease, produces paræsthesiæ and convulsive movements in the groups of muscles which it represents. Destruction of it causes not only a paralysis, but a certain amount of cutaneous anæsthesia.

The various sensori-motor centres are not sharply limited, but lap one over the other, so that the motor area for the forearm, for example, extends over somewhat into that for the shoulder. The corresponding sensory areas are more diffuse, so that it takes a much more extensive destruction of a certain area of the motor cortex to produce an anæsthesia of the arm than it does to produce a paralysis of the arm.

The sensori-motor area including some adjacent parts is called by Flechsig the "somatosphere," because here he thinks are received the afferent impulses of general (visceral) as well as special tactile and muscular sensations from all over the body.

Bilateral Representation.—Those muscles of the two sides of the body which act together have a double representation in the brain. For example, each group of muscles used in inspiration has a centre in both hemispheres; consequently, when one centre is destroyed no paralysis results, for the reason that the other centre continues its work. In the same way some of the muscles of the face, such as those for closing the eyes, have a double representation, and a lesion destroying the centre for the orbicularis palpebrarum on one side will not usually cause paralysis, because of the continued action of the centre of the other side. The more perfect and habitual the associated action of the muscles of the two sides of the body, the more completely can one centre do the work of its associate. The best examples of the muscles having the double representation are the orbicularis palpebrarum, the muscles of the vocal cords, the muscles concerned in deglutition and in respiration. The muscles of the viscera and blood-vessels have no known representation in the cortex of the human brain.

The special sensations have a bilateral representation also; but the more specialized the sense the less can one hemisphere take the place of the other.

Occipital, Parietal, and Temporal Lobes—Centres of Special Sense.—The special senses have two centres—the primary and the secondary. The primary centres are connected with the ganglia at the base of the brain; the secondary centres are situated in the cortex.

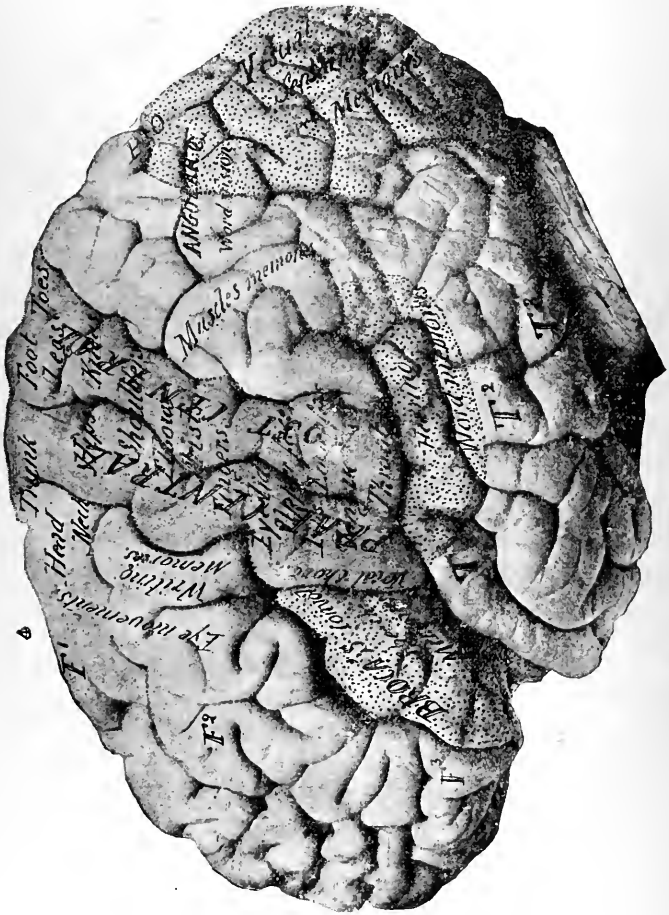


FIG. 191.—THE LOCALIZATION OF THE FUNCTIONS OF THE CONVOLUTIONS OF THE BRAIN.

The *primary centre for vision* is in the posterior part of the optic thalamus, the external geniculate body, and anterior corpora quadrigemina. The secondary centre is situated in the occipital lobe, and particularly upon its mesial surface and in that of the cuneus, known as the calcarine fissure. Each occipital lobe is the centre for visual impulses from the corresponding half of the retina of each eye; for example, the left occipital lobe is the centre for vision of

the left half of the retina of each eye. This relation is shown in the diagram (see Optic Nerve). Total destruction of both occipital lobes, or even of a considerable part of them if the destruction in-

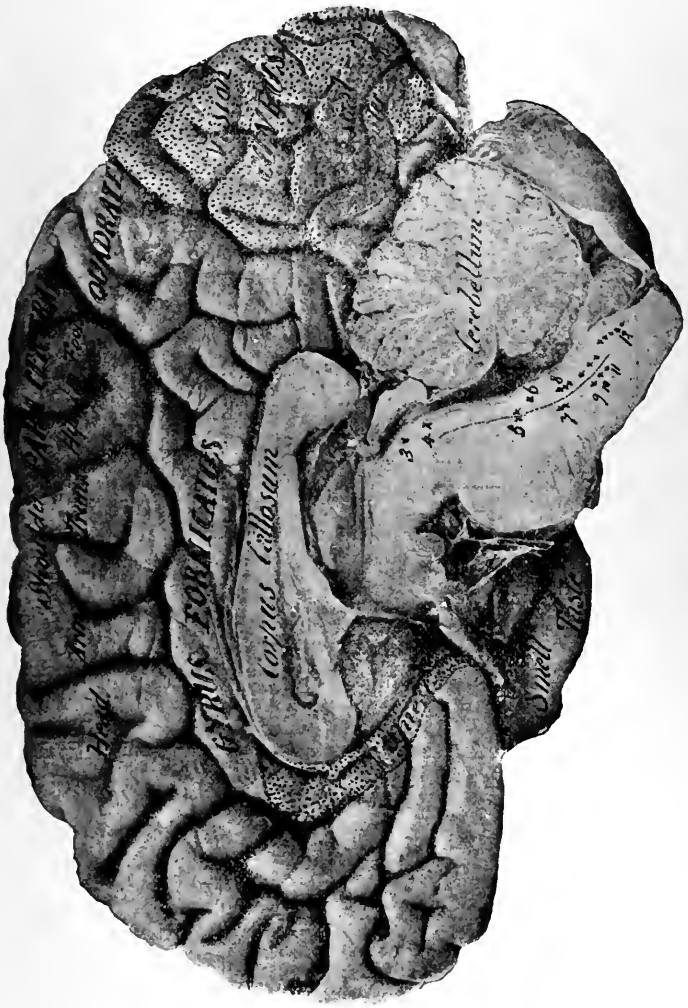


FIG. 192.—THE LOCALIZATION OF THE FUNCTIONS OF THE CONVOLUTIONS OF THE BRAIN.

volves the median surface, will cause blindness. Destruction of one lobe causes only half-blindness or hemianopsia.

The *primary centre for hearing* is in the posterior tubercle of the corpora quadrigemina and the internal geniculate body. The secondary centre is in the cortex of the first and second convolutions of

the temporal lobe. Destruction of one temporal lobe causes deafness in the opposite ear. This deafness, however, is not complete because the sense of hearing has a bilateral representation; each ear, in other words, sends fibres to the temporal lobes of each side, although more fibres cross over than go to the lobe of the corresponding side. The consequence is that the loss of one temporal lobe is in a measure supplied by the other (*vide* Aphasia).

The *primary centre for smell* is in the olfactory lobes. The secondary centre is probably in the anterior part of the limbic lobe, the uncus and in part of the hippocampal convolution. Whether the tracts for the sense of smell are connected with the optic thalamus or other ganglia is not definitely known.

The primary centre for taste is not known, but sensations of taste may connect with the optic thalamus before passing into the secondary centre, which is in the hippocampal convolution.

Centres for Memories.—There are certain classes of sensations and perceptions, simple in character and frequently repeated, so that they finally get to be used almost automatically in their work. These impressions relate to the use of the muscles in speech, in writing, and in gesture language; also to other frequently repeated purposeful movements of the limbs. The muscular movements in writing and speaking are so often repeated that certain areas in the cortex are set apart for the memories of these processes, memory being simply a revival of previously registered impressions. The visual sensations and the ideas elaborated from them, which are frequently repeated in learning to read, have also a centre which is set apart for them. This forms a centre for the visual memories of language. In the same way there are auditory sensations and ideas elaborated and so frequently repeated as to be used automatically in acquiring language. These are stored up as auditory memories. We have what may be called motor memories connected with speech and gesture. These special memories have been found to have a certain localization in the brain. The centre for the memories of the articular movements of speech is in the posterior part of the third left frontal convolution; the centre for the memories of the movements of writing is not perfectly well known, but is thought to be at the posterior part of the second left frontal convolution. The centre for the memories of gesture language is unknown. The centre for the memories of ordinary co-ordinate movements is probably in the inferior parietal lobule. The centre for the visual memories of written language is in the angular gyrus, extending backward from there into the occipital lobe. The centre for the auditory memories of spoken language is in the posterior part of the

first and the corresponding upper part of the second temporal convolution. In right-handed people all the memory centres are in the left cerebral hemisphere; in left-handed people they are in the right hemisphere. The destruction of these memory centres produces different forms of aphasia, as will be described later. In addition to that, disturbances in these centres are produced by lesions which cut off the associating fibres connecting these centres with each other or with motor or sensory centres proper.

The Centrum Ovale, Corpus Callosum, and the Associative Functions of the Brain.—The different parts and centres of the brain are connected together by the associating tracts and with lower levels by the projection fibres. The simpler and less developed centres of the two halves of the brain are closely connected by fibres that run chiefly in the corpus callosum. The more highly specialized and less simple in function a centre, the less close is its commissural connection and the more independent is one half of the brain from the other. Thus the centres for the movements of the thorax in respiration are closely bound with each other; those for the purposeful movements of the hands less so; those for receiving visual impressions are almost independent; and the centres for the memories, which are still more highly specialized, are practically entirely independent. We infer that the higher mental functions, therefore, work either in one cerebral hemisphere or in the other, and that the two halves of the brain do not co-operate with each other in much of the higher intellectual work.

The corpus callosum is the great commissural tract connecting the two cerebral hemispheres and their respective centres. The anterior commissure does some of the same work, being more specially connected with the function of olfaction. The posterior commissure has comparatively few bilateral connecting fibres, its function being more to connect the thalamus with the cranial nerve nuclei and other centres below.

The Corpus Striatum.—This ganglion is in close relation with the cerebellum and with nuclei in the pons. It is also in connection with fibres that come up from the muscle-sense tract, in the spinal cord. Its functions are therefore probably connected with securing co-ordinate and purposeful movements. Destruction, however, of this ganglion in the human brain produces no definite symptoms, and local lesions of it cannot be diagnosticated. It is therefore called clinically a *latent region*.

The Thalamus Opticus.—The thalamus is in relation by its projection fibres with the frontal, parietal, occipital, and temporal cortex. The fibres that go to the occipital cortex are connected

with the optic tract, and have to do with the function of vision. The fibres that go to the temporal lobe are connected with the auditory tract, and have to do with the function of hearing. The optic thalami seem to have some relation to the expression of emotions. In cerebral paralyses in which they are involved the patient cannot involuntarily express joy, grief, etc. Lesions of the posterior part of the thalamus will produce partial blindness. Other than this, lesions of the optic thalamus produce no definite symptoms which enable us to make a local diagnosis. Disturbances of hearing have not certainly been traced to lesions in the thalamus. It is probably a primary centre for sensations of touch, muscular sense, and perhaps for smell and taste, but no definite facts in human pathology have as yet satisfactorily proved this. Lesions of the thalamus sometimes produce various forms of mobile spasm, but these are generally attributed to irritation of the fibres of the internal capsule, which go close to it. Hence, aside from disturbances of vision, the optic thalamus also must be considered clinically a latent region.

The Corpora Quadrigemina.—The anterior tubercles of the corpora quadrigemina, together with the external geniculate bodies, form part of the primary centres of vision. The anterior tubercles, however, have to do chiefly with reflex movements of the pupil and the ciliary muscles. The posterior tubercles of the corpora quadrigemina and the internal geniculate body are connected with the auditory nerve, and have to do with reflex movements associated with hearing and space sensations. They also appear to receive some fibres from the cerebellum; their injury or disease produces some disturbances in equilibrium and possibly in hearing. Owing to the fact that the nuclei of the third nerves and the red nuclei lie beneath the corpora quadrigemina, lesions of these latter produce irritations and paralyses of the third nerve, disturbances in equilibrium, and forced movements. Lesions in this neighborhood sometimes cause somnolent and stuporous states.

The *red nuclei* are connected with the anterior cerebellar peduncles on the one hand and with the lenticular nucleus and optic thalamus on the other, and are concerned in securing equilibrium and the adjustment of the body in space.

The Cerebellum.—The cerebellum is connected with the pons, the cerebrum, and spinal cord. It sends impulses down into the antero-lateral columns of the cord, and through the anterior peduncles to the red nuclei, the thalamus, corpora striata, and the central convolutions. It receives impulses from the cortex of the frontal lobes, which go down into the pons, connect with nuclei

there, and thence pass up into its hemispheres (indirect motor tract). It also receives impulses from the spinal cord, through its peduncles, which go on to the thalami and brain cortex (indirect sensory tract). There is therefore a nervous circuit between the cerebrum, brain axis, cerebellum, and spinal cord. The cerebellum has thus the function of securing the higher automatic and pyscho-reflex movements, and through its further relations with the space-sense nerve (eighth) of enabling us to keep our equilibrium and maintain our relations in space. The vermis or median lobe is the part which in man is most important in doing this work. Lesions of the lateral lobes or hemispheres produce few direct symptoms, and they are called latent regions. Injuries of the median lobe, however, produce disturbances in equilibrium, forced movements, and a peculiar form of inco-ordination in gait which is known as cerebellar ataxia. Lesions of the middle peduncles produce forced movements also, the forced movements being either toward or away from the side of the lesion, according as it is an irritating one or a destructive one.

The *pons Varolii* contains some of the cranial nerve nuclei and collections of nerve cells which are connected with fibres from the cerebral cortex on the one hand and the cerebellum on the other. It also contains the long tracts of nerve fibres that pass from the cerebrum down through into the medulla and spinal cord and transverse tracts of fibres which connect the two hemispheres of the cerebellum. Lesions in it cause disturbances in function of the cranial nerves and of the motor, sensory, and commissural tracts.

The *medulla oblongata* contains centres of the cranial nerves, and in it also are various reflex and automatic centres controlling and regulating the vasomotor system, respiratory and cardiac rhythm, visceral movements and secretion.

The *olivary bodies* are connected with the cerebellum, basal ganglia, and with the spinal cord. When injured, disturbances of equilibrium and co-ordination occur.

The Latent Regions of the Brain.—There are certain parts of the cerebral cortex destruction of which and irritation of which produce no special and distinctive phenomena in man. These are the greater part of the temporal lobe of the right side and a portion of the temporal lobe on the left side. A part of the inferior parietal lobule also may be regarded as a latent region. The frontal lobe we have already spoken of as being concerned with certain mental functions, but lesions here often produce no symptoms, and may be to a certain extent regarded as latent. These latent regions are called by Flechsig the higher or associative centres. The corpora

striata, optic thalami, portions of the centrum ovale, and the two lateral hemispheres of the cerebellum are latent areas.

Brain Weight.—The average weight of the male brain is 1,358 gm.; that of the female, 1,235 gm. The weight varies with age, sex, race, and intelligence, and with a number of other factors. The average weight of the brain at birth is 327.8 gm.; the brain grows rapidly until the age of four, then more slowly until the age of seven, then very slowly up to the age of sixteen to twenty. At about the age of forty-five in man and fifty in woman it begins to lose weight slowly, and at the age of eighty or over it has lost about 120 gm. (4 oz). The brain of man weighs absolutely about nine per cent more than that of woman. Relatively to the body weight, the brain weight of man is about 2 per cent; that of woman a very little less. The sexual difference is extremely small.*

The brain weighs more in the civilized races, and more in certain of the civilized races than others; the brains of English, German, and Scotch weigh more than those of French, Italian, and Russian. Some of the African and Australian tribes have the smallest brain, the average negro brain weighing 1,250 gm. When a brain weighs less than 1,130 gm. in man or 990 gm. in woman, it is called a microcephalic brain; if the weight is above 1,490 gm. in man or 1,345 gm. in woman, it is called a megalocephalic brain.

Brain weight has a certain relation to intelligence, which is not, however, an absolute one. Among a hundred men of more than average intelligence, the percentage of large brains would be about 25, whereas the percentage of large brains among persons of ordinary or low intelligence would be not more than 4 or 5. In estimating the importance of brain weight, one must consider the height, the weight or volume of body, muscular mass, and superficial area; these are called the somatic factors. The following formula has been devised by Snell for estimating the mental power of different animals·

$$P = \frac{H}{K^S}$$

In this formula P represents the psychical factor or the amount of intelligence, H the brain weight, K the body weight, S the somatic factor. The somatic factor has been estimated to be for mammals about 0.666. Applying this formula, we find that, expressed relatively, the intelligence of man equals 0.87; woman, 0.86; the ape, 0.42; the rabbit, 0.59; the birds from 0.167 to 0.09.

The relative weight of different parts of the brain is about as follows: frontal lobes, 28 per cent; parietal lobes, 36 per cent; occipital, 10 per cent; temporal, 13 per cent; lobus caudicus or island of Reil, 9 per cent; pons, 1½ per cent. The cerebellum weighs about one-eighth as much as the cerebrum. The proportion of the gray to the white matter in adults is 60 to 40 (Vierordt).

*J. C. Brown finds that after making all allowances, woman's brain weighs about one ounce less than man's.

The depth of the primary fissures is not quite an inch (20 to 23 mm.).

There are from one thousand two hundred to two thousand million cells in the cerebrum, and about ten million large cells in the cerebellum (Meynert).

About one million cells to a square centimetre is the estimate of Engel.

Preserving and Cutting the Brain.

The brain should be placed in a gallon of a 2½-per-cent solution of bichromate of potassium. This must be changed daily for a week, then twice weekly for a fortnight; then it should remain in the solution for three or four months, a few crystals of thymol being added. After about three months place the brain in 95-per-cent alcohol. In a few days it will be ready for cutting. Or for permanent or temporary use the brain may be placed in a 4-per-cent solution of formalin. Later it may be changed to alcohol or Müller's fluid.

In cutting the fresh or preserved brain for the purpose of locating gross lesions, remove the pons, medulla, and cerebellum, place

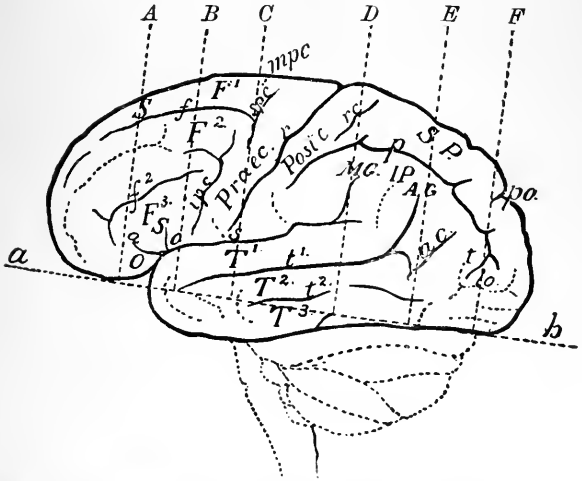


Fig. 193.—Showing the Points at which the Sections are Made. *a*, *b*, Horizontal line from base of frontal to base of occipital lobe; *A*, vertical section through middle of third frontal convolution; *B*, through operculum, *o*; *C*, through superior precentral and lower end of Rolandic fissures; *D*, through *rc* and posterior end of Sylvian fissures; *E*, through angular gyrus and anterior occipital fissure; *F*, through parieto-occipital fissure. *B* should be about half-way between *A* and *C*; *E* half-way between *D* and *F*. (See Figs. 191-196.)

the brain on its base, and make sections in accordance with the directions (Fig. 193). The sectional views exposed are shown in the following series of cuts, which are based upon those of Exner.

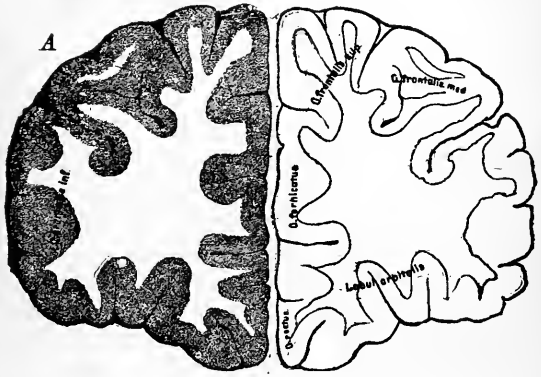


FIG. 194.—SECTION THROUGH LINE *A*, FIG. 193.

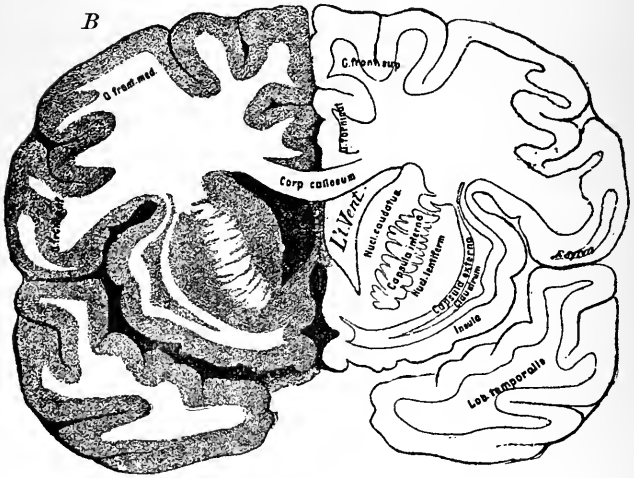


FIG. 195.—SECTION THROUGH LINE *B*, FIG. 193.

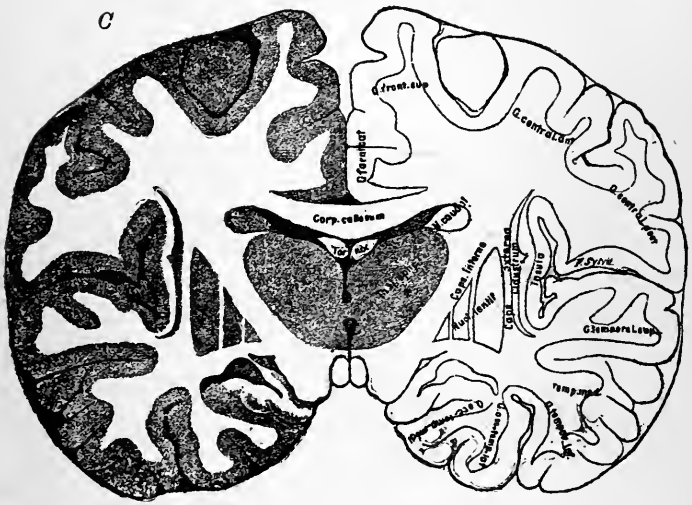


FIG. 196.—SECTION THROUGH LINE *C*, FIG. 193.

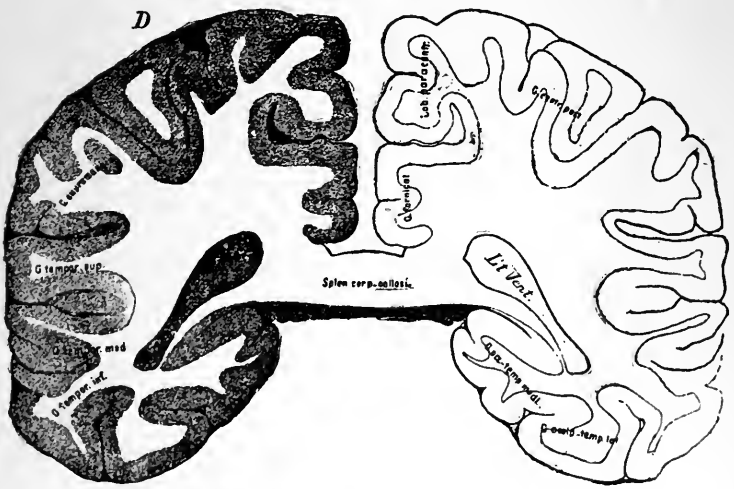


FIG. 197.—SECTION THROUGH LINE *D*, FIG. 193.

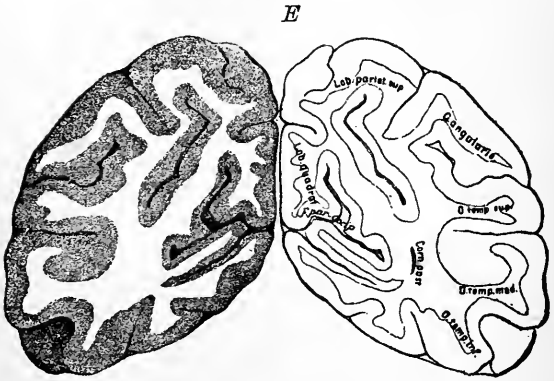


FIG. 198.—SECTION THROUGH LINE *E*, FIG. 193.

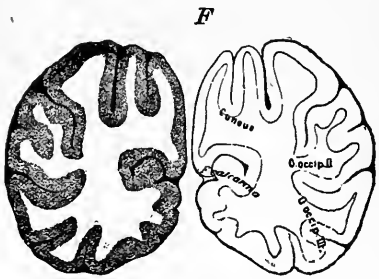


FIG. 199.—SECTION THROUGH LINE *F*, FIG. 193.

CHAPTER XVIII.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

General Symptoms.

It will add to the intelligibility of descriptions of brain diseases and their symptoms if one first makes himself familiar with certain general symptoms that underlie more or less nearly all organic disorders of this organ. Symptoms due to disease of the brain may be placed in four classes: first, general symptoms of brain irritation; second, general symptoms of brain presssure; third, symptoms of focal irritation or destruction; and, last, those due directly to the pathological process itself.

The symptoms of brain irritation are headache, vertigo, vomiting, photophobia, mental irritability, insomnia, peculiar feelings of fulness and pressure about the head, noises in the ears or in the head, tenderness about the scalp, and in severe cases convulsive symptoms and delirium.

The symptoms of brain compression are headache, vomiting, mental hebetude or dulness, perhaps some form of paralysis, contracted pupils, and eventually coma. With this there are often constipation and retracted abdomen.

The symptoms of brain irritation are often, perhaps usually, associated with a hyperæmia. The symptoms of brain compression may be associated with anæmia or œdema, and often in states of malnutrition in which the brain is impoverished the symptoms resemble much those of compression. Pressure symptoms and irritation symptoms often lap one into the other and they cannot always be sharply distinguished.

Focal symptoms depend almost entirely upon the location of the particular lesion. If it is in a motor area, focal symptoms of irritation would be spasmodic phenomena, such as convulsions. If the lesion were destructive, the symptoms would be those of paralysis or anæsthesia.

The symptoms due directly to the pathological process itself may be very slight. Thus in case of a tumor of the brain the symptoms are mainly caused by pressure, irritation, and local disturbance of certain special parts of the brain. In suppuration, however, the proc-

ess itself may produce general symptoms such as are associated usually with sepsis—chills, irregular fever, mental hebetude, prostration, emaciation, and sweats.

Among the symptoms produced by focal lesions there are a few which deserve some preliminary general study, because they may be caused by lesions of very different kinds and occur consequently in very different forms of diseases. Those symptoms which we wish particularly to study here are hemiplegia and aphasia. These represent the two great dominating symptoms pertaining on the one hand to motor disturbance and on the other hand to sensori-motor disturbance.

Hemiplegia.—Hemiplegia is a paralysis of one half of the body involving the side opposite the lesion. The face, arm, and leg are usually all paralyzed; the arm most, the leg next, the face least. Hemiplegia may be either acute in onset or slow and progressive. Acute hemiplegia is the result usually of hemorrhages and softenings of the brain, more rarely of inflammations and injuries. Progressive hemiplegia begins gradually, as its name implies, and slowly increases until the height of the disease is reached. It is usually caused by tumors growing in one side of the brain, but it may be caused by a slowly developing patch of sclerosis, which sclerosis may be in turn only a part of a multiple sclerosis. Further description of the peculiarities of hemiplegia will be given under the head of Special Diseases of the Brain.

Aphasia.—Aphasia is a disorder of the faculty of language; and it has a number of varieties, in accordance with the particular part of the brain involved and the particular portion of the mechanism of this faculty that is destroyed. By the faculty of language we include the processes by which we hear, see, and at the same time appreciate the meaning of symbols. It includes also the faculty of expressing to others by voice, writing, or gesture our ideas. It has therefore a receptive side and an emissive side. We may have lesions in the brain which destroy that part of the language faculty concerned in our power of seeing and understanding written words or the gesture language. In reading understandingly one sees certain words; these words revive certain visual memories connected with past perceptions. Thus one sees the word "book;" this suggests to him past memories of form, color, tactile and other sensations associated with the past perceptions of books. There is a certain centre in the brain where these visual memories for letters and words are located. When this centre is destroyed the memories are destroyed and the word "book" or any other written word conveys no meaning. The patient can spell out the

letters, he can see the letters, but he cannot read any more than if he had never been taught. The condition is known as *alexia* or *word blindness*. Again a person may have learned to associate certain gestures with definite ideas, as the motion of carrying a glass to the mouth with that of drinking, or the motions of using a knife and fork with that of eating, or the motions of the deaf-and-dumb alphabet with certain words and ideas. These memories of gesture language are located in certain regions, and when they are destroyed the patient is no longer able to understand gestures or the sign language. This condition is known as sign blindness. When a person is not able to understand the significance or uses of things about him, he has *apraxia*. Apraxia, sign blindness, and alexia all come under the general head of *mind blindness*, because, though the patient can see, he does not understand what he sees. A person hears certain words, as, for example, the word "knife." This conveys to him a certain idea of the form, color, and other properties associated with knife. The memories associated with the auditory perception of different words are stored up in a certain locality which is the centre for auditory memories. When this centre is destroyed the person hears spoken words, but they convey to him no meaning. All that is said to him sounds as if it were in a foreign language: he hears, but he does not understand. This condition is known as *word deafness*. So much for the receptive or sensory side of language.

In communicating our ideas, we speak, write, and make gestures. In speaking we make use of the organs of articulation, and this use involves the fine adjustment of a delicate muscular apparatus. In the act of expressing ideas we have to bring into play the memories of the past muscular movements of this articulatory mechanism. These movements were learned by a slow and painful process during infancy. After the power of speech is acquired, the mechanism works readily and almost automatically, because we only have to send a stimulus to the centre which presides over the stored-up memories of the impulses to innervate properly the mechanism of speech. There is, therefore, a centre for the memories of the movements of articulation—a centre which is of course closely connected with the motor areas that directly innervate the larynx, pharynx, and oral and facial muscles. When a lesion destroys this centre for speech memories, a person is unable to reproduce the words necessary for expressing an idea; for example, he sees a knife, he knows what it is, but the memory of the motions necessary to express the word "knife" is gone. To him it seems that the name is gone, and that is the common way of expressing it.

He cannot say the word "knife." The patient may wish to express the idea of pain. He feels the pain, he knows that he has pain, but he cannot revive those motor memories which are concerned in expressing the word "pain;" he cannot tell, therefore, in words what is the matter with him. When a person is thus troubled, he is said to have a form of motor aphasia for which the particular name given is *aphemia*. In the same way there is a centre for the memories of the muscular movements concerned in writing; and when a lesion destroys this centre the patient is unable to write, though he may be able to speak. This condition is called *agraphia*. There is a centre, less well defined, for the memories of the movements used in gesture language, and when this is destroyed the person is unable to express his ideas by gesture or sign language. This condition is known as *amimia*. In some cases, patients are able to speak and write, but they skip words, repeat often, and talk confusedly. There is here a lesion of the tracts associating the language centres, and the condition is called *conduction aphasia*, while to his stumbling speech the term *paraphasia* is given.

In attempting to classify these various aphasic conditions we group together as much as possible those symptoms which we know are related to rather definite areas of the brain. The divisions are based on symptoms, yet each symptom group has an anatomical seat which in many cases can be exactly determined.

The following are the principal forms of aphasia:

Auditory aphasia.
Motor aphasia, { aphemia. / agraphia.
Visual aphasia.
Conduction aphasia.
Mixed aphasia.

Each of these forms has certain subdivisions of which the analysis and recognition are matters of great interest, but I shall only suggest the lines along which such investigations are pursued. The excessive use of diagrams has filled up literature with exceptional cases and impaired the clearness and sense of proportion with which the clinical pictures of aphasia should be presented. No doubt, however, the ultimate result will be useful.

In the examination of a case of aphasia, the following twelve questions should always be put to the patient:

1. Can he hear sounds?
2. Can he hear spoken words?
3. Can he understand the words spoken?
4. Can he see objects?

5. Can he see words written or printed, and read them silently?
6. Can he understand written or printed words, *i.e.*, can he read intelligently?
7. Can he speak voluntarily?
8. Can he repeat words?
9. Can he read aloud?
10. Can he write voluntarily?
11. Can he write to dictation?
12. Can he copy?

In *auditory aphasia*, the principal symptom is that the patient has word deafness. He is unable to understand spoken language, though he hears the sounds and is not at all deaf. The lesion is in the first and second temporal convolutions of the left hemisphere. When the lesion is extensive, the patient has many other aphasic symptoms, because all speech centres are closely united functionally. Visual, auditory, and articulatory memories are brought into play together, and form a kind of internal language circuit around which the nerve impulses play in the production of speech. If now a patient has an auditory aphasia with deep and extensive injury of the temporal lobe, it will be found that he cannot understand spoken words, neither can he read intelligently. He cannot repeat words or read aloud, and he cannot write to dictation, nor copy. He can speak voluntarily, however, but he skips words and is paraphasic. This form is called cortical sensory aphasia (Wernicke). If the lesion is smaller, or if the case improves and the injured tissue to some extent heals, it will be found that the patient still has word deafness and cannot repeat words or write to dictation; but he can talk and read and write voluntarily. This forms a subcortical aphasia.

In *motor aphasia* or aphemia the principal symptom is that the patient cannot speak voluntarily, he cannot repeat words or read aloud. He cannot write voluntarily or to dictation, but he can copy. He hears, sees, understands both written and spoken language. This is the most common type of aphasia and its seat is known to be in Broca's convolution, *i.e.*, the third left frontal. In its completer type as given above it is called cortical motor aphasia, but this means nothing. In severe cases, the patient cannot read understandingly except to a limited extent and the power of understanding spoken words is also impaired. On the other hand, in lighter forms the patient can read and write and understand, and has lost only the power of voluntary speech, of repeating words and reading out loud.

Agraphia is a symptom of nearly all the forms of aphasia. It

is oftenest seen in aphemia and is most complete in this type. There is no form of aphasia, however, in which agraphia is the only symptom, and the evidence of a writing or graphic centre in the cortex is not proven, though it is probable.

Visual aphasia is accompanied by an inability to read words understandingly, though the patient can see them. He is able to speak and understand spoken words. Alexia is thus the characteristic symptom. There are often hemianopsia and some hemiataxia or anæsthesia. Two principal forms have been described. In one there is considerable agraphia, as well as inability to read either silently or aloud. Here the lesion involves the cortex of the angular gyrus and supramarginal lobule (cortical alexia). In the other form, the patient has a pure alexia, and can write though he cannot copy. There is usually hemianopsia present. The lesion here is in the subcortical substance of the angular gyrus.

Conduction and Mixed Aphasia.—There are very few cases of pure conduction aphasia. When it occurs there is paraphasia and paragraphia; the patient repeats words over and over in a kind of verbal intoxication, or mixes things so that the speech is almost gibberish. Still he can express himself and can write, read, and understand. The lesion is usually in the island of Reil or the convolutions about the fissure of Sylvius.

Practically conduction aphasia is usually mixed with a visual or auditory aphasia.

Malformations of the Brain and its Envelopes.

Congenital malformations of the brain are of little practical importance, for in most cases the monsters cannot live and in all cases they are better dead. I shall simply give a brief enumeration of the important forms.

Abnormalities of the brain.	Anencephaly. Micrencephaly and microcephaly. Porencephaly. Absences or malformations of parts, *e.g.*, cyclopia.
Abnormalities of brain and its envelopes.	Acrania. Meningocele. Encephalocele. Hydrencephalocele.

Anencephaly is always present with acrania. In anencephaly the cerebellum and part of the basal ganglia may be present. In such case the child can live a short time (Fig. 200).

Micrencephaly and Microcephaly.—Micrencephaly is a condition in which the brain is only partially developed. If, as is usually the case, the cranium is also abnormally small, it is called microcephaly. It is due, probably, in all cases to an inherent defect in the growth

of the brain. Virchow has asserted, however, that there is a form in which the abnormality is caused by a premature growing together

FIG. 200.—ACRANIA.

of the cranial bones, a micrencephaly being a result of the mechanical condition. An adult cranium whose gross circumference meas-

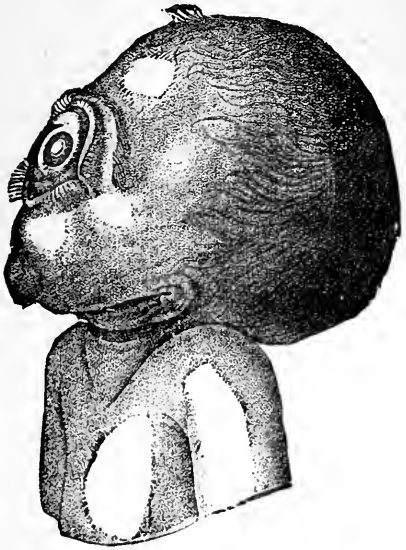


FIG. 201.—CYCLOPIA.

ures less than 43 cm. will contain a micrencephalic brain. The normal minimum weight of the adult brain is 960 grams for man

and 880 for woman. It should bear the ratio to the body at birth of 14 per cent, and of 2.37 per cent in adult life (Vierordt).

Porencephaly is often an artificial condition. It will be described later.

In cyclopia there is an undivided anterior cerebral vesicle; the orbits form a continuous cavity with a single rudimentary eye (Fig. 201).

Meningocele is a hernia of the brain membranes, arachnoid, and dura mater through a cleft in the skull. In encephalocele the brain also protrudes. Both these forms occur usually in the occipital region and almost invariably in the median line. In hydrencephalocele there is a sac with fluid contents.

Diseases of the Membranes of the Brain.

The diseases to be considered under this head are anæmia and hyperæmia, inflammation of the dura mater or pachymeningitis, and inflammation of the pia mater or leptomeningitis.

Anæmia and Hyperæmia of the Membranes of the Brain.—Anæmia of the membranes of the brain is a condition that cannot be separated from anæmia of the brain substance, and will be considered in connection with it. Hyperæmia of the brain membrane, so far as it relates to hyperæmia of the pia mater, must also be considered in connection with hyperæmia of the brain tissue. Dural hyperæmia, or congestion of the dura mater, is a condition which occurs as the result of injuries, sunstroke, and of certain infective poisons, especially that of syphilis. The symptoms are those of pachymeningitis of the slight grade, and will be described under that head. They consist mainly of pain, occasional attacks of vertigo, and sensations of fulness about the head. The treatment is that for the beginning stages of a meningitis.

Inflammation of the Dura Mater or Pachymeningitis Externa.—It has been the custom to describe two forms of pachymeningitis, the external and the internal. Internal pachymeningitis, or hæmatoma of the dura mater, is properly a hemorrhagic disorder, and is described under the head of Dural Hemorrhages. A true inflammation confined to the internal surface of the dura alone is of extremely rare occurrence.

Pachymeningitis externa is a disease that involves, at first at least, the outer surface of the dura, and is usually of surgical origin and interest.

Etiology.—Accidents, injuries, caries of the petrous bone in mastoid disease, of the ethmoid bone in ozæna, necrosis, syphilis, and erysipelas are the usual causes.

The *symptoms* are local headache, fever, delirium, sometimes even convulsions and paralysis. In the severe cases the disease has usually extended and involved the pia. Pus is generally formed, and burrows between the bone and dura. The disease is recognized mainly by the discovery of the local cause.

The *course* is acute or subacute.

The *treatment* is a surgical one.

Inflammation of the Pia Mater, or Leptomeningitis.—Inflammation of the pia mater has the following types: simple meningitis due to some infection, epidemic cerebro-spinal meningitis due to a specific general infection, tuberculous meningitis, serous meningitis, and syphilitic meningitis.

Most of all these forms of meningitis may be either acute or chronic, the chronic form being usually simply a sequela of the acute.

Acute Simple Leptomeningitis—*Etiology.*—Acute leptomeningitis is always due to an infective process reaching the cerebral membranes usually directly from without, but sometimes through the blood. Trauma, and especially acute alcoholism predispose to this. The most common source of infection is disease of the middle ear and mastoid cells. Disease of the frontal sinuses and upper nasal passages; operations on those parts; disease, injuries, and fractures of the cranial bones—are also common causes. Pneumonia is the most frequent infective disease in which the pyogenic organisms are carried by the blood. After this come pyæmia, septicæmia, variola, scarlet fever, more rarely endocarditis, empyema, rheumatism, measles, typhoid fever, and mumps. Occasionally a brain abscess reaches the surface and sets up a meningitis. Insolation can of itself not cause it. The disease is more frequent in males, and is distributed through all ages of life, though it occurs oftener in the young.

Symptoms.—The symptoms in the various types differ somewhat, but have a general similarity. They are to be broadly grouped into the prodromal, the irritative, the depressive, and the paralytic stages.

Prodromal symptoms are shorter and less marked in simple meningitis than in tubercular. The patient suffers from malaise, languor, headache, vertigo, irritability, loss of appetite, and vomiting. Of these symptoms headache is the most notable.

In the second stage the dominant symptoms are headache, delirium, rigidity of the neck, hyperæsthesia of the skin, retraction of the abdomen, vomiting, irregular fever, contracted and often unequal pupils, sometimes optic neuritis or retinitis. The headache

is usually persistent, with exacerbations of great intensity. Rather early in this disease the patient's mind begins to wander; he mutters incoherently; he may have periods of violence alternating with stupor. In some cases there is a continuous low muttering delirium. Vomiting also occurs early and is of a violent, explosive (projectile) character. This symptom is not always present. The head is bent back and the patient can be lifted from the pillow by placing the hand under the occiput. There is sometimes a general rigidity which resembles catalepsy. Drawing a dull point along the skin causes a red line to appear (*tache cérébrale*). Pinching or rubbing the skin causes much pain. The abdomen falls in and assumes a characteristic "boat shape." The pupils are usually contracted and uneven. The eyes are intolerant of light. Optic neuritis occurs often when the inflammation is at the base, but it is a late symptom. Convulsions and local paralyses of the cranial nerves, causing slight strabismus, ptosis, or facial palsy, may occur. The fever is irregular in course and not high—101° to 103°. The pulse is usually irregular or rather intermittent. It varies greatly in frequency and may be rather slow—50 to 70. Respiration is rather quickened and sometimes irregular. The bowels are constipated; the urine is small in amount and sometimes albuminous.

In the paralytic stage the patient becomes stupid or comatose; there is still some rigidity, except in the very last stages. The abdomen is still greatly retracted, the pupils may now dilate, the skin become moist, and the patient's bowels and bladder move involuntarily. Death then occurs in one or two days as a rule.

When the disease is mainly on the convexity of the hemispheres there are more delirium, convulsive and paralytic troubles; when confined to the base there is less delirium, while paralysis of cranial nerves, optic neuritis, vomiting, and retraction of the head are commoner or more prominent.

Course and Duration.—The disease may begin suddenly, and the patient pass at once into the comatose state, dying in a few days. Usually the process lasts one or two weeks; it may be prolonged for several weeks.

The *prognosis* is very grave, but it is less serious than in tuberculous meningitis and more serious than in the cerebro-spinal form.

The *diagnosis* is based on the presence of an exciting cause, such as disease of the ear or nose, trauma, infective fevers, and upon the presence of the symptoms given. It is usually easily recognized, the main difficulty being to distinguish it from tuberculous and cerebro-spinal meningitis.

To assist in diagnosis it is permissible to make a lumbar puncture, draw off the fluid, and examine it for bacteria.

Pathology.—The disease is a fibro-purulent or purulent inflammation. It involves usually the base more than the convexity, but the reverse may happen. The ventricles are often involved and may be independently inflamed. There are descriptions, therefore, of simple basilar meningitis, meningitis of the convexity, and ventricular meningitis or ependymitis. The inflammatory deposits are most conspicuous along the course of the Sylvian fissure and the vessels branching from it, about the optic chiasm, and at the posterior and under surface of the cerebellum and the sides of the pons. It may lie only in the subarachnoid cavity, but usually the arachnoid and sometimes the dura are implicated. There is increase of fluid in the ventricles and arachnoid cavities, and this fluid may be turbid. The surface of the ventricles may show an inflammatory process.

The micro-organisms found in meningitis are the pneumococcus, streptococcus pyogenes, intracellular diplococcus, the pneumo-bacillus, and a bacillus resembling that of typhoid fever. Still others have been described, and the process is apparently a mixed infection, though the pneumococcus is found oftenest.

Treatment.—Prophylaxis is the most important measure, as there is no specific treatment. Chronic disease of the ear and nasal sinuses should be attended to, and injuries of the skull treated with the strictest regard to antisepsis. The patient should be kept quiet, a dose of calomel given, and small doses of iodide of potassium administered at frequent intervals. An ice cap may be applied to the head and hot applications to the feet. Hot poultices along the upper spine are useful. Opium must be given for the pain, if needed; and antipyretics or phenacetin sometimes answer, in a measure. The internal use of iodoform has been highly recommended, gr. vi. to gr. xij. daily; shaving the head and rubbing upon it an ointment containing twenty per cent iodoform, then covering the scalp with an oiled-silk cap, is a treatment highly spoken of. Surgical intervention is sometimes justifiable.

Epidemic Cerebro-Spinal Meningitis (Spotted Fever).—This is an acute infective disorder and is produced by a special micro-organism. It has certain peculiar clinical characteristics which lead us to describe it separately. Anatomically the changes involve the spinal membranes as well as the cerebral.

Etiology.—The disease most frequently attacks children, but it may occur at any age. Males are affected rather more often than females. It prevails in the form of epidemics which affect cold and temperate climates especially, and which travel from one part of the country to another. It may occur sporadically. It most frequently develops during the winter season, and attacks persons who are

living in crowded houses, tenements, or barracks. It is slightly contagious. One attack does not confer an immunity against a second.

Symptoms.—The general appearance of a person attacked with the disease is that of one who has been poisoned by some agent which is extremely prostrating to the whole system and at the same time one which has a specific inflammatory effect upon the meninges of the brain and spinal cord. When the disease is rapid and malignant, the patient seems to die of an acute toxæmia before any inflammatory process has time to develop. In milder cases and those of longer duration the prostration is less, and the evidences of inflammation of the meninges then develop in the typical way.

The disease may begin with prodromal symptoms of malaise, discomfort, pain in the neck, vomiting, and headache. As it develops, the headache, accompanied by giddiness, increases, pain and stiffness in the neck become more marked, pains run down the back and radiate to the limbs; there is photophobia, and delirium in many cases is present. The skin is hyperæsthetic; the pulse rises to 120 or higher; the temperature varies very much and is usually raised to 103°, 104°, or even more. The bowels are generally constipated. In most cases there develop certain skin eruptions, usually in the form of purpuric spots; herpes, urticaria, and erythema are occasionally seen. These eruptions vary very much in different epidemics; the purpuric spots are the most important from a diagnostic point of view, and have given to the disease the name of *spotted fever.* As the disease progresses the symptoms of irritation and pain give way to those of somnolence, stupor, and paralysis. Optic neuritis, acoustic neuritis, and inflammation of other cranial nerves take place, and paralyses of the limbs may be added.

The disease may run a short and malignant course, killing the person in a few hours or one or two days. In moderate cases it lasts about two weeks. A large number of different varieties of the disease are described, such as the abortive form, fulminating form, and typhoid form. The disorder is often complicated with pneumonia and bronchitis, less often with inflammation of the joints and serous membranes. The disease often leaves very serious sequelæ, the most important being deafness and spinal irritation or chronic spinal meningitis. A large number of deaf mutes owe their affliction to this disease.

Pathological Anatomy.—In the very acute cases the post-mortem shows nothing but the evidence of very severe blood-poisoning. In the milder and more chronic cases an inflammation involving the pia and arachnoid of the brain and cord is found. This inflamma-

tion is fibrinous or fibro-purulent in character, and may be accompanied with the exudation of a good deal of inflammatory material. Bacteriological researches show that this disease is due to the presence of a specific micro-organism which is apparently very much like that which causes pneumonia.

The *diagnosis* is based upon the history of an epidemic of the disease being present, upon the presence of the ordinary symptoms of acute cerebral and spinal meningitis, such as headache, delirium, retraction of the head, the sunken abdomen, hyperæsthesia, and pains; finally, the presence of the peculiar purpuric spots or of herpes of the face will enable one to make a positive diagnosis. One must learn to distinguish the disease from typhus, tetanus, uræmia, pneumonia, and from the other forms of meningitis, especially the tuberculous. The diagnosis is often made difficult by the fact that cerebro-spinal meningitis may occur in a sporadic form, and it is well known that after a community has been once visited by an epidemic these sporadic cases are apt to crop up from time to time for many subsequent years. The sudden onset of the disease, the spinal symptoms, the skin eruption, the absence of history of injury or of evidence of tuberculosis will usually enable one to recognize the disorder. Lumbar puncture may be used.

The *prognosis* varies much with the epidemic, but the disease is always a serious one. The mortality ranges from twenty to eighty per cent; it is worse when the disease comes on suddenly and severely, with early coma. It is better in persons over the age of ten. Cranial-nerve complications are unfavorable, in that they are apt to leave permanent deafness. Severe spinal complications are apt to leave their mark in the form of a chronic meningeal trouble.

Treatment.—There is no specific remedy for the disease, and the ordinary antiphlogistic measures such as mercury and iodides are of less value than in other forms of meningitis. The patient should be given sustaining food, and everything possible should be done to counteract the depressing effects of the toxæmia. Opium or morphine internally, chloral, digitalis, quinine, benzoate of sodium and salicylate of sodium, and alcohol are the drugs which have been specially recommended. Warm baths, hot moist applications, and leeches have all been tried with more or less good results.

Tuberculous Meningitis (Acute Hydrocephalus).—This is a form of meningitis due to infection with the bacillus tuberculosis. It differs pathologically from other forms in the character of the infective organism; anatomically, in the fact that the inflammation is usually and chiefly basilar and never purely purulent; etiologi-

cally, in that it chiefly affects young children; and symptomatologically, in the presence of prodromata and a more irregular course.

Etiology.—Tuberculous meningitis occurs chiefly between the ages of two and ten, sometimes in infancy, rarely in adult life, very rarely after the age of fifty. Males are rather more subject to it. A hereditary history of phthisis, a scrofulous diathesis, bad hygienic surroundings, and the presence of tuberculosis elsewhere in the body predispose to it. Tuberculous milk, the eruptive fevers, especially measles, blows on the head, and great emotional excitement appear to act as exciting causes.

Symptoms.—A knowledge of the prodromal symptoms is especially important. These are paroxysmal and intensely severe headaches and darting pains in the head, vertigo, loss of appetite, explosive vomiting without nausea, the vomited matter being usually colorless and watery, constipation, an altered disposition, and irritability. The *tache cérébrale* or cerebral macule, more rarely ptosis and facial paralysis may appear early. The prodromal stage often lasts, with remissions, three or four weeks. When the disease sets in there is more persistent headache; vomiting, fever, and the other symptoms of meningitis already described appear. The irritative stage gradually passes into the paralytic and comatose. Death occurs in two or three weeks. In infants the disease often runs a very obscure course, the patient showing chiefly symptoms of brain compression.

Pathological Anatomy.—In rapidly fatal cases, with severe symptoms, there may be only an intense congestion of the brain with numerous miliary tubercles in the pia mater at the base and over the convexity. Here we must assume that a bacillary toxin causes the symptoms. In most cases there are decided deposits of tubercles at the base, with fibrinous inflammatory deposits about the optic chiasm, along the fissure of Sylvius, at the sides of the pons, and elsewhere. Miliary tubercles are seen scattered over the convexity and in the choroid plexus and ventricles. They are generally found in the spinal membranes also, especially over the cauda equina. The tubercles lie beneath the pia surrounding the small vessels. They may coalesce into large tuberculous nodules. There is usually an increase in the arachnoid fluid, and in most cases an increase in the ventricular fluid. Somewhat rarely there are very great distention of the ventricles and compression of the convolutions. This condition used to be called *acute hydrocephalus*. Small spots of softening may be seen from obliteration of the vessels by the tubercles. The bacillus tuberculosis is found in the tuberculous nodules.

Diagnosis.—As regards the form of the disease, this is based on the hereditary history, the age, the existence of tuberculosis of the lungs or other organs, and the peculiar prodromata of the disease. Occasionally tubercles can be seen on the choroid. Lumbar puncture of the spinal canal with withdrawal of fluid and its examination for bacillus is a method of diagnosis which may be tried.

Prognosis.—This is usually absolutely bad, yet post-mortem observation of patients dying with practically no inflammatory change makes it seem possible that the disease might be checked, and a good many cases are reported in which it apparently has been done. Some of these are, however, probably cases of hereditary syphilis.

Treatment.—So far as is now known, this is not different from that given under the head of meningitis elsewhere. It seems, however, as if in time some antitoxin may be discovered which will check the progress of the poison and the development of the tubercle; meanwhile the best thing to do is to give small doses of iodide of potassium at frequent intervals and use symptomatic treatment.

Chronic Hydrocephalus.

This is a disease mainly of infancy, characterized by a gradual enlargement of the head, with mental deficiency and symptoms of brain irritation caused by an accumulation of fluid in the ventricles of the brain.

The old term, "acute hydrocephalus," meant an acute inflammation with effusion, but the name is not needed and is best dropped. Chronic hydrocephalus is not an inflammatory process, but one due to mechanical causes or to defects in structure or nutrition. The fluid always accumulates in the ventricles of the brain; hence chronic hydrocephalus is always *internal*. The so-called external forms of hydrocephalus are inflammatory or else are secondary to meningeal hemorrhage or brain atrophy. Chronic hydrocephalus is almost always a disease of infancy and is generally congenital. It may, however, be acquired. In speaking of chronic hydrocephalus, we refer to the chronic internal congenital disease.

Etiology.—Four out of five cases begin at birth or within the first six months of life. Syphilis (J. Lewis Smith), alcoholism, lead poisoning in the parents, and some unknown family taint predispose to the disease. Poverty and poor nutrition and rickets are also factors.

Symptoms.—The head may be so large at birth that instrumental help is needed. More often the parents notice a gradual increase in the size of the child's head, beginning soon after birth. The

forehead bulges, the occiput stands out, the fontanelles and sutures widen, and pressure shows evidence of fluctuation. Meanwhile the face does not grow much and the result is to give a triangular shape to the head. It may measure twenty-four, twenty-seven and one-half (Minot), thirty-two (Bright), and even forty-three inches (Klein) in diameter. These extreme measurements are reached only after one or two years. With this abnormal growth of the head, mental and physical symptoms appear. The infant is restless and irritable; its appetite may be good, but the general nutrition is poor and its bodily growth is retarded. The mind does not develop; usually it does not or cannot learn to walk. It may be unable to support the weight of its head. There is strabismus and

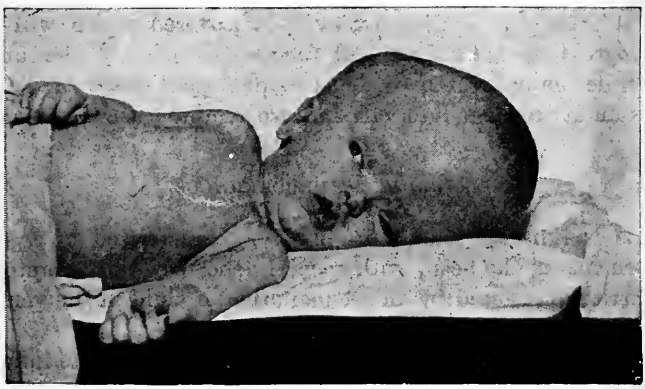


FIG. 202.—CHRONIC HYDROCEPHALUS.

sometimes optic atrophy. The pressure of the dropsy thins the orbital bones and forces down the axis of the eyeballs (see Fig. 202). Vomiting, coma, and convulsions eventually appear, and the child dies of exhaustion or some intercurrent disease in two or three years.

In some cases the trouble is less serious, it ceases to progress, the bones solidify, and the child grows up with good intelligence.

Chronic hydrocephalus sometimes develops in late childhood and in adult life. It is then due to some tumor or inflammatory process obstructing the venæ Galeni and the aqueduct of Sylvius. The symptoms are chiefly those of brain pressure, and the disease cannot be recognized with certainty.

The hydrocephalus which is associated with the brain atrophy of insanity and old age or with general dropsical conditions has no kinship with the process we are now describing.

Pathology.—The disease is due to the gradual accumulation of

a serous fluid in the ventricles of the brain. The cause of this is an inflammatory or developmental obliteration of the aqueduct of Sylvius or the foramen of Magendie and the adjacent lateral foramina of Mierziejewski. This prevents the escape of the ventricular fluid into the general arachnoid cavity. Contributing factors are congenital or acquired defect in the absorbents of the ventricles and a rachitic and easily yielding skull. The attempts to make chronic hydrocephalus an inflammation are failures, though there is at times a thickening of the lining membrane of the ventricles and other changes of a syphilitic character. The lateral ventricles are principally and often solely affected, and these are so distended as to press out their cerebral walls, flattening the convolutions, and turning them into a thin shell often less than a quarter of an inch in thickness. Sometimes only one lateral ventricle, and in rare cases only the fourth ventricle, is affected by the dropsy.

The *diagnosis* has to be made from rickets and an acute inflammatory process. In rickets the head is square, the fontanelle does not bulge, the enlargement is less, and there are signs of the disease in the bones elsewhere.

Prognosis.—The congenital cases usually result fatally in a few months, or at least before the third year. Those developing in infancy may persist for four to six years; and in mild cases the disease ceases toprogress and a fairly healthy adult life is reached.

Treatment.—A great many measures have been recommended, but there is no unanimity about any one of them. In such a state of therapeutics it is safe to say that treatment is of little use. The most rational measure is the inunction of mercury and the administration of iodide of potassium combined with tonics. Surgical measures, such as tapping the ventricles, are irrational and need not be discussed. Quincke's method of tapping the spinal canal will not apply in these cases, though it is a practicable measure, as I have found by experiments on the cadaver. Strapping the head with diachylon plaster is recommended by Trousseau and by J. Lewis Smith.

Alcoholic Meningitis (Serous Meningitis, "Wet-Brain").

Alcoholic meningitis is a clinical term used to indicate the peculiar group of cerebral symptoms which is seen in persons who have succumbed to the effects of prolonged alcoholic intoxication. The disease is not a true meningitis but an acute toxæmia of the brain with serous effusion, and may be called, for the purpose of convenience, a serous meningitis.

Etiology.—The disease occurs oftenest in men simply because of the more frequent indulgence of the male sex in alcohol. It rarely develops until a person has been drinking eight or ten years, and, consequently, affects people oftenest between the ages of thirty and forty. The exciting cause is commonly alcohol and in this country whiskey or what are known as "hard drinks," but beer and ale will accomplish the same result. I have rarely seen the disease in wine drinkers. The persistent use of morphine, cocaine, and chloral may lead to much the same condition. The exciting cause is usually a continuous drinking bout of two or three weeks, ending in delirium tremens. The delirium tremens, however, is not by any means always present. The patient may pass from a condition of prolonged intoxication into the condition of alcoholic meningitis or "wet-brain."

Symptoms.—In case delirium tremens has occurred, the patient after two or three days of prolonged delirious excitement gradually sinks into a semi-coma. This is accompanied by a muttering delirium. The patient is sufficiently conscious to have flitting delusions and hallucinations of sight and hearing. At this time he is able to drink and take food; the pulse is rather rapid, the temperature is usually normal or may be raised one-half or one degree. The skin is hyperæsthetic and pressure upon the muscles of the arms or legs or abdomen causes pain. The pupils are usually rather small. Often at this time conjunctivitis and keratitis may appear. After a few days the patient's stupor becomes deeper and he can be aroused only with difficulty. The arms and legs are now somewhat stiff, the reflexes are exaggerated, the neck is stiff and slightly retracted, and attempts to move it cause expressions of pain. The abdomen is retracted and the skin and muscles are still very hyperæsthetic. The lids are closed. The pupils are small and do not react well to light. The tongue is coated and usually dry, and urine and fæces may be passed involuntarily. The patient may linger this way for several days more. The pulse becomes rapid and feeble, the extremities are stiff and cold. The skin is dry and loses its elasticity, so that when pulled up between the fingers it stays in folds. "Putty skin" is a good name for this. The coma deepens, the temperature may rise to 103° or 104°, and symptoms of pneumonia may appear as the scene closes. On the other hand in some cases the patient does not pass into the worst stage, the mind becomes clearer, the hyperæsthesia lessens, food is taken better, and the bowels are moved voluntarily. Improvement continues and in three or four weeks the convalescence begins.

Pathological Anatomy.—I have made autopsies and careful micro-

scopical examinations in over twenty cases of the character just described. In nearly all the brain is found to be somewhat pale, the arachnoid contains two or three ounces of serous fluid, the subarachnoid space is saturated with fluid, and the ventricles are dilated. Sections through the brain sometimes show punctate hemorrhages and in rare cases spots of hemorrhagic extravasation are seen surrounded by softening. Occasionally the beginning of a suppurative cerebral meningitis will be seen. Microscopic examination shows in the uncomplicated cases that there is no true inflammatory process. There is often congestion, but not always; the commoner condition is an œdema of the brain tissue, the perivascular and pericellular spaces being dilated. The nerve cells show various stages of degeneration, not pigmentary in character, however. The chromophilic granules are often unstained, or, if stained, have lost their true relations, and seem broken down. The cell outlines are irregular; the nucleus lies near the periphery of the cell, and in some cases has broken out and escaped from it. Sometimes there is a large number of neuroglia cells in the pericellular spaces. The disease is undoubtedly, primarily at least, a toxæmia not due directly to the influence of alcohol but to the poisons which have developed in the body as a result of the condition of inanition and the paralysis of the digestive function caused by the prolonged ingestion of alcohol and abstinence from food. The cell degeneration is more like that which is known as "degeneration from a distance," such as is seen in nerve cells when the neuraxon is destroyed. It is a degeneration which affects especially the body of the cell and not so much the nucleus; hence the remarkable power of recovery from this condition which so many people show.

The *diagnosis* of the disease is to be made from ordinary suppurative meningitis, from acute serous meningitis due to infection, and from acute encephalitis. In most cases the history of the patient is quite sufficient to establish the diagnosis. The symptoms of themselves are almost identical with those of ordinary acute suppurative meningitis. The only distinctions which I have been able to observe are that in suppurative meningitis there is more fever, there is less of the low delirium, hallucinations are rare, and there is an earlier and more profound coma. In other words, it is an acuter and more severe malady than alcoholic meningitis. The absence of convulsions and paralysis and the presence of hyperæsthesia, rigidity, and contracted pupils, as well as the absence of pyrexia, are usually sufficient to distinguish the disorder from encephalitis or encephalitis complicated by alcoholic meningitis.

The *prognosis* is bad when the disease has become well developed

and when decided coma and rigidity have set in. A prognostic criterium which I have long used and which is fairly accurate is this: if the patient has not a stiff neck he will get well, but when stiff neck comes on the patient dies.

The *treatment* of the disorder should be instituted at the very beginning. If there are still any relics of the debauch, as shown in the condition of the stomach or intestinal tract, the stomach should be washed out and, at all events, a thorough purge should be given. The patient should then be fed most liberally with hot milk given every two hours; beef tea and an egg beaten up in milk may also be given, but the condition of practical starvation on the part of the patient should always be borne in mind. Stimulants in the shape of whiskey should not be administered if it is possible to avoid it, but strychnine in doses of one-sixtieth of a grain every two hours is often useful. An ice cap should be applied to the head and at times leeches or large blisters seem to be useful applied to the back of the neck. The patient, however, should not be much depleted. When he becomes comatose it means that the ventricles and arachnoid cavities are becoming filled with water. At this time tapping the spinal cord may be tried. I have done this in a number of cases and have at times removed two or three ounces of fluid with some amelioration of the symptoms and never any bad results, but the measure has never been tried early on promising cases and I have never seen it do any permanent good.

CHAPTER XIX.

DISEASES OF THE BRAIN.

These diseases, like those of other parts of the nervous system, consist of malformations, vascular disturbances, inflammations, softenings, hemorrhages, degenerations and scleroses, chronic infections, tumors, and functional disorders.

Cerebral hyperæmia is a condition in which there is an excessive amount of blood in the cranial cavity; it may be acute or chronic, active or passive.

Etiology.—In the description which is to follow I shall refer only to those conditions of hyperæmia of the brain which are pathological. It is a well-recognized fact that hyperæmia of the brain occurs physiologically under excitement and overactivity of the heart and from various stimuli; but a pathological condition of acute congestion may be induced by sunstroke, certain drugs such as alcohol, and by injuries; also by mechanical causes which prevent the exit of the blood from the cranium. An acute congestion also occurs in mania and in many forms of fevers, as well as in the initial stage of meningitis. A chronic cerebral hyperæmia may be induced by the causes already mentioned as bringing on acute congestion. The prolonged use of alcohol, prolonged mental excitement, overwork, and worry may also lead to this condition. The foregoing causes lead to what is known as active congestion, in which the blood is driven in excess into the brain through the arteries. A passive congestion may exist in which the blood is prevented from leaving the brain and is kept mainly in the intracranial veins. The causes of passive congestion are chiefly mechanical, such as cardiac disease and mechanical obstructions about the neck from tight clothes, and an obstruction to the flow of blood from the lungs by playing on wind instruments.

Symptoms.—A great deal has been written regarding the symptomatology of cerebral hyperæmia, but many of the statements made are nothing but guesswork. Probably the main symptoms produced by an active congestion of the brain are a sense of fulness

and pressure, a feeling of constriction about the head, some headache which may be vertical, mental excitement or irritability, confusion of ideas, vertigo, insomnia, ringing in the ears, and pulsating sounds in the head. These symptoms are sometimes increased when the patient lies down, and are generally increased when the patient bends the head over so as to prevent the return flow of blood from the brain. It is impossible to diagnosticate passive hyperæmia from active through the symptoms alone, but probably in the former condition the disturbances and symptoms mentioned are less marked; in other words, an active hyperæmia produces more phenomena than passive. In either case examinations of the fundus of the eye and of the tympanum furnish no sure criteria.

Pathology.—Cerebral hyperæmia used to be regarded as nearly synonymous with cerebral neurasthenia. In the writer's opinion, it is secondary to the neurasthenic state, and is produced, if it exists in that state, by the impaired vasomotor innervation which is characteristic of neurasthenia. It is not wise, therefore, to make the diagnosis of cerebral hyperæmia often as the primary condition. It is only after traumatisms and sunstroke or after a meningitis that we can speak of the cerebral hyperæmia as being in a certain sense the primary condition to be treated. The statement made by some writers that cerebral hyperæmia underlies certain conditions of acute delirium, of aphasia, of paralysis, and even dementia or insanity can hardly be supported. In many of the cases of cerebral hyperæmia, in which symptoms are produced, there is undoubtedly a condition of toxæmia which is a contributing factor to most of the symptoms.

Treatment.—The specific treatment of cerebral hyperæmia, when indicated, consists in giving large doses of fluid extract of ergot and bromide of potassium. One or two drachms of the ergot three times a day and fifteen or twenty grains of bromide of potassium may be prescribed. Wet cups to the back of the neck, the cautery in the same region, ice caps, purgatives, quiet and rest, and a careful regulation of the diet and the bowels are all important measures.

Cerebral Anæmia.—This condition, like hyperæmia, may be either acute or chronic.

It occurs among the young; more often in females than in males. It is seen in early adult life, when it is induced by the various causes producing general anæmia, and again after the climacteric, when it is due to organic changes in the cerebral arteries of the nature of an obliterating endarteritis. Bright's disease and syphilis, exhausting diseases and profuse hemorrhages, and such disorders of digestion and nutrition as lead to general anæmia produce

also cerebral anæmia. A potent cause of acute cerebral anæmia is fright.

Symptoms.—The symptoms of acute cerebral anæmia are vertigo, confusion of ideas, nausea, faintness, or complete syncope. In chronic cerebral anæmia the symptoms are mental apathy and a feeling of disinclination to work, tendency to somnolence in the daytime and insomnia at night, mental depression, headaches which are usually frontal or vertical, occasionally some vertigo and tinnitus. There may be spots before the eyes and undue sensitiveness to sounds. In children some very severe symptoms are attributed to cerebral anæmia, but here, as in hyperæmia, it is probable that there are other causes at work, particularly toxic agents or reflex disturbances.

Diagnosis.—A chronic anæmia of the brain can hardly be recognized except through the evidences of a general anæmia. When this is present and there are also symptoms such as have been described, a fairly certain diagnosis can be made. We must look upon cerebral anæmia as being in almost all cases a secondary phenomenon, except in the aged, and then the trouble is due not alone to poverty of the blood, but to the fact that the circulatory apparatus is diseased. It is generally believed that in cerebral anæmia the symptoms improve somewhat by the horizontal position and are made worse by the upright position. It is also asserted that in anæmia the pupils rather tend to be dilated, while in hyperæmia they are contracted.

Treatment.—Treatment should be directed toward enriching the supply of blood and toward improving the general nutrition. It consists, therefore, in the administration of preparations of iron and of such tonics as the mineral acids, strychnine, quinine, and nitroglycerin.

INFLAMMATION OF THE BRAIN.

The forms of acute inflammation of the brain are acute suppurative encephalitis or brain abscess, acute exudative encephalitis with hemorrhage, and acute polio-encephalitis. The only important form of chronic inflammation is multiple sclerosis.

Acute Suppurative Encephalitis (Abscess of the Brain).

Brain abscess is a suppurative inflammation which affects the parenchymatous and other structures of the organ. It is always a focal disease, but may be single or multiple.

Etiology.—The primary cause of all forms of brain abscess is a microbic infection. The form of microbe, its mode of entrance, and the part of the brain attacked vary greatly. The predisposing causes relate chiefly to age and sex. Brain abscess rarely occurs before the first year or after the fiftieth year of life. It is rather frequent in young people, and occurs on the whole oftenest between the ages of ten and thirty. Males are more often affected than females in the ratio of about three to one. The exciting causes are chiefly disease of the ear, of the nose, and of the cranial bones, injuries, and remote suppurative processes.* To this may be added infectious fevers and the presence of tumors. Chronic inflammation of the middle and internal ear is the most common cause of brain abscess, especially when that disease affects the tympanum and mastoid cells. Caries of the ethmoid and nasal bones and of the orbital cavity leads to brain abscesses in a considerable proportion of cases. After chronic ear and bone diseases injuries are the most frequent cause. The injury may be a compound fracture with direct infection from the open wound, or the abscess may be the result of *contrecoup* and may develop in a part of the brain opposite to that which was injured, or the abscess may develop below the point injured, there being apparently healthy tissue between the surface of the brain and the diseased part. These abscesses develop through laceration of brain tissue and subsequent infection of the wound with organisms. The most common remote suppurative processes which are followed by brain abscess are tuberculous inflammation of the lungs, fetid bronchitis, and empyema. Brain abscess may develop, however, from distant points of suppuration on the extremities or in almost any part of the body. Pyæmia may lead to the production of brain abscess. Among the infectious fevers which are complicated with brain abscess are diphtheria, typhoid and typhus fevers, erysipelas, small-pox, the grippe. The oidium albicans or thrush may also be a cause. Brain tumors sometimes become surrounded by a suppurative encephalitis or may break down with the formation of mixed suppurative and neoplastic tissue. Tuberculous tumors are most frequently accompanied by suppurative encephalitis.

Symptoms.—Brain abscesses take sometimes an acute and sometimes a chronic course. In acute cases the symptoms develop rapidly and the disease runs its course in a few days or weeks. The symptoms come under the general head of those of pressure, those

* In nine thousand consecutive autopsies at Guy's Hospital there were fifty-seven brain abscesses due to ear disease and one due to nasal disease (Pitt).

of poisoning from the diseased focus, and local symptoms due to irritation or destruction of certain special areas of the brain. The pressure symptoms are those of headache which is often very severe and persistent, vomiting which is quite frequent though not invariable, vertigo, and a condition of mental dulness which may pass into a delirium ending finally in coma. Optic neuritis often occurs. The pupils are apt to be irregular, but furnish no definite indications. The pulse is usually slow, ranging from 60 to 70, but it varies a great deal. The temperature is normal or subnormal as a rule, but this also varies, and it may rise several degrees above normal, always running an irregular course. The toxic symptoms are those which we get in septic poisoning; namely, prostration, irregular fever, emaciation, anorexia, and such mental and sensory disturbances as have already been referred to. As a result of local irritation or destruction, there occur convulsions, paralysis, aphasia, and disorders of some of the special senses. Convulsions are not very common. When they occur they are generally of an epileptic character. The paralysis is usually in the form of hemiplegia. The cranial nerves are not often involved, if we except the optic. The urine is said to show a diminution in chlorides and an increase in phosphates. The patient dies finally in coma from exhaustion.

In the chronic form of brain abscess the symptoms may for weeks, months, or years remain practically latent, after the exciting cause has been at work and after the abscess has been established. The patient during this latent stage may suffer from headache, vertigo, mental irritability, and depression; he may at times have a convulsive attack. Occasionally there will be an exacerbation of the disease, at which time he suffers from intense pain, vomiting, perhaps delirium or a convulsion. From this he recovers and continues in a fairly good state of health again. After a variable period, usually of weeks or months, the terminal stage sets in. This terminal stage of the chronic form may assume very much the characters of the acute form already described. In other cases it shows itself by a sudden apoplectic or epileptic seizure or a sudden attack of coma, in which the patient sinks and rapidly dies. These terminal phenomena are due to the fact that the abscess, which has been previously encysted and quiescent, suddenly breaks into a lateral ventricle or through the surface of the brain, or to the fact that a hemorrhage occurs into the abscess.

Complications.—The common complications of brain abscess are a phlebitis of the superior petrosal and lateral sinuses and a meningitis. The phlebitis accompanies abscesses that are caused by disease of the ear. The meningitis may be caused by ear dis-

ease, but more frequently accompanies abscesses due to injury. When phlebitis is present there will be found an œdema about the ear and neck and a hardness of the jugular veins. In meningitis there is apt to be more rigidity of the neck, more pain, and there are often cranial-nerve paralyses.

Pathology. — Acute suppurative encephalitis resembles acute suppurative myelitis in the intimate nature of the changes that take place. There is an intense congestion of the parts, which gives it a reddened appearance and which used to give to this process the name of *red softening*. This condition, however, is only the initial stage of the suppurative inflammation and does not deserve to be ranked as a special form of inflammatory process. It is possible that in some cases the inflammation may get no farther than the stage of red softening. The congestion then gradually disappears, absorption of exudate occurs, and a more or less complete recovery takes place. When the process continues, however, the parts become crowded with leucocytes and infiltrated with inflammatory exudate. The nerve fibres and cells are destroyed, in part mechanically, in part by the poisonous influence of the pyogenic organisms. The nerve cells lose their normal contours, swell up, and disintegrate; the neuroglia cells absorb the broken-down detritus and swell up, forming what are known as granular corpuscles; the leucocytes increase until a purulent mass is formed. The total result is a mixture of broken-down nerve fibres and cells, leucocytes, and granular bodies. Bacteriological tests show the presence of various pyogenic microbes. The abscess thus formed varies in size from one centimetre to six or eight centimetres in diameter (two-fifths of an inch to three inches). It is generally somewhat round, and if the case is chronic a fibrous wall is formed. It takes from three to four weeks for such a wall to develop (Fig. 203). Brain abscesses are usually single, occasionally there are two or three. In some conditions they are multiple; that is to say, there may be fifteen, twenty, or more. Multiple brain abscesses are always small and are usually due to pyæmic infection.

Location.—Brain abscesses involve the cerebrum oftener than the cerebellum, in the proportion of about four to one (Barr). They occur rather oftener in the right cerebrum. They are very rare in the pons and medulla. The cerebral lobes oftenest affected are the temporal and frontal. In the cerebellum it is the lateral hemispheres that are most frequently attacked. The seat of the abscess has important relations to the cause. Abscesses due to ear disease are almost always either in the temporal lobe or in the cerebellum. If the ear disease is in the tympanum, the cerebrum is usually the

seat of the abscess. If the disease is in the mastoid cells, the cerebellum is usually the part affected. If the disease is in the labyrinth, the abscess is also more apt to be in the cerebellum. This distribution of the seat of the disease is due to the anatomical relations of the bony parts to the temporal lobe and cerebellum, respectively. Brain abscesses due to injuries are more frequent in the frontal and temporal lobes. What are known as idiopathic brain abscesses—that is, those which arise without any known cause—are

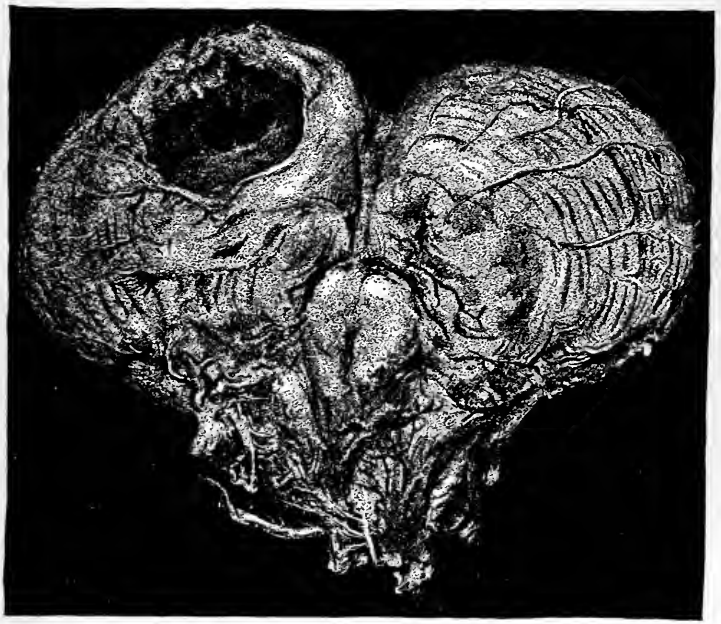


FIG. 203.—ABSCESS OF CEREBELLUM.

most frequent in the frontal lobes. This is because most such cases are due to an unrecognized affection of the nasal cavities and ethmoid bone. Brain abscesses due to suppurative processes in the lungs and pleura are probably embolic; and, as the emboli are carried up into the middle cerebral artery, the brain abscesses having this origin are situated in the field supplied by this artery. In children under ten, in whom brain abscess is usually due to ear disease, the cerebellum is more apt to be affected.

Course.—Acute abscesses last from five to fourteen days, rarely over thirty days. Traumatic cases run the shortest course. Chronic abscesses may have a latent period of weeks, months, and in rare cases even one or two years. When terminal symptoms come on death occurs in a few days. In a few cases brain abscesses

have been spontaneously evacuated through the nose. Aside from this, the termination is always a fatal one unless surgical interference takes place. There is sometimes a recurrence of the abscess after an operation.

Diagnosis.—The diagnosis of brain abscess is based upon the history of injury, aural or nasal disease, remote suppuration, upon the general symptoms of sepsis, upon the presence of headache, vomiting, slow pulse, normal or subnormal and irregular temperature, a local tenderness of the scalp and rise of temperature over

FIG. 204.—SHOWING THE POINTS WHERE THE TREPHINE IS USUALLY APPLIED AND THE RELATIONS OF THE SINUS. The divisions on the lines indicate quarter-inches (*Lancet*).

the seat of the lesion, hebetude, delirium, optic neuritis, rapid wasting, and diminution of chlorides in the urine.

The diagnosis of the location of the abscess is based upon the history of its cause, whether from injury, ear disease, emboli from the lungs, or nasal disease; also upon the presence of hemiplegia, local convulsions, tenderness and rise of temperature of a certain area of the scalp. As brain abscesses are apt to affect latent regions like the temporal and frontal lobes, local diagnosis is usually difficult. The diagnosis must be made from tumors of the brain, meningitis, and phlebitis of the sinuses. The differential points are given under the heads of the diseases mentioned. The presence of leucocytosis helps in distinguishing the presence of suppuration.

The *prognosis* of the disease is absolutely unfavorable unless some surgical interference is resorted to. The few rare cases of spontaneous evacuation of the abscess would not lead to any practical modification of this statement.

Treatment.—The actual treatment of a brain abscess after it has developed is, as already stated, exclusively a surgical one. The successes so far have not been very great, but they have been sufficient to justify operation and to furnish greater hope for the future, when a more exact diagnosis can be made and a wider surgical experience has been obtained. The accompanying figure shows the points to be located in trephining for abscess from ear disease. Something is due to the patient in the way of prevention, especially in cases of persons who have chronic aural or nasal disease with carious processes. These should be carefully watched and treated.

Acute Exudative Encephalitis of the Gray Matter (Polio-Encephalitis).

Acute exudative polio-encephalitis is a disease which affects the gray matter forming the cranial nerve nuclei. It has two forms, polio-encephalitis superior, and inferior.

Acute polio-encephalitis inferior is a disease which is strictly analogous in course to acute polio-myelitis. The special symptoms simply depend upon the peculiar location of the disease. They consist of an acute glosso-labio-laryngeal palsy, and are referred to under the description of bulbar paralysis.

Acute polio-encephalitis superior is a disease in which the nuclei of the nerves supplying the eye muscles are involved; it is also a disease analogous to polio-myelitis anterior, and has been described under the head of ophthalmoplegia.

Acute cortical polio-encephalitis of children is a disease which, according to Strümpell, involves the gray matter of the convexity of one of the cerebral hemispheres. It also is supposed to be strictly analogous to an inflammation of the anterior horns of the spinal cord. The real existence of this disease is still doubtful.

Acute Exudative Encephalitis with Hemorrhage (Hemorrhagic Encephalitis).

This is a form of encephalitis affecting diffusely various areas of the brain and characterized by intense congestion with capillary hemorrhages followed by inflammation, never ending in suppuration and sometimes terminating in partial or complete recovery.

Etiology.—One of the principal exciting causes of this disease is probably the infection of influenza, but it also occurs in connection with other infectious fevers, such as typhoid, typhus, and epidemic cerebro-spinal meningitis. It has been known to follow malignant endocarditis and it occurs in connection with the puer-

peral state. I have seen a number of cases occurring as a result of acute alcoholism, although it is not impossible that there is a coincident infection. In about one out of ten autopsies on the brain of persons who have died from acute alcoholism, I have found large foci of hemorrhagic softening with evidence of inflammatory reaction about it. The disease occurs also after sunstroke. Acute hemorrhagic encephalitis, when due to infection, occurs most frequently in the young, that is to say, in persons under twenty, and more often in females than in males. Probably the increased susceptibility of the young to the infectious fevers is the explanation of this.

Symptoms.—The disease begins rather suddenly and without notable premonitory symptoms. The patient is seized by headache followed by fever sometimes reaching 105° F. This may be associated with vertigo, vomiting, photophobia, and delirium. The symptoms of irritation disappear and are followed by a condition of semicoma or stupor. The patient can generally be partly aroused, and he does not have the stiff neck or the small pupils of meningitis. The respirations are shallow and frequent, the pulse is rapid and feeble. As the disease progresses, the deep reflexes are diminished and later the sphincters may be involved. After the patient has lain in a semicomatose condition for several days, he may become less stupid and more irritable and restless; or after two or three weeks of comparative stupor he may begin gradually to improve and, in a few weeks more, convalescence takes place.

In some cases an epileptic convulsion may occur in the early part of the disease. Again, as the disease develops, aphasia and paralysis of the arm or leg, or hemiplegia, may appear. In accordance with the location of the inflammation, the patient may have disturbances in the motor sphere, or he may have hemianopsia, hemiataxia, or disturbance of the cranial nerves, such as nystagmus, or eye palsy, or difficulty in speech and deglutition. An optic neuritis may also occur.

Course and Prognosis.—The disease is always serious but is not by any means always fatal. It may in its milder form run a course of two or three weeks, the patient gradually coming out of his stupor and making a slow recovery. In other cases the coma continues to deepen and the patient dies of exhaustion, and in still other cases the disease passes into a chronic state in which he lingers for weeks and even months.

Diagnosis.—The disease in the young is probably more often mistaken for meningitis. It is to be differentiated from this by the sudden onset with coma, the absence of projectile vomiting, pin-

hole pupils, stiff neck, hyperæsthesia, and rigidity of the limbs. The presence of hemiplegia or local paralysis, or the occurrence of an epileptoid attack, would point to encephalitis. The diagnosis from meningitis, however, cannot always be made. The previous occurrence of an attack of grippe in the young, or of exposure to the sun or acute alcoholism in the adult, would lead to the probability of an encephalitis, provided the symptoms of meningeal irritation could be excluded. Tuberculous meningitis can be excluded usually by the presence of premonitory symptoms and the absence of any tuberculous focus in the lungs or intestines. By the help of lumbar puncture fluid can be withdrawn from the spinal sac, and examination of it might be of service in excluding at least tuberculous meningitis or a serous meningitis.

Pathological Anatomy.—The pathological process underlying this disease consists of an acute inflammation with intense congestion and numerous small hemorrhages and capillary emboli. There is some hemorrhagic exudation as well as infiltration of leucocytes, with a certain amount of softening of the cerebral tissue in the neighborhood. The parts most frequently affected are the semi-ovale, the temporal lobes, the base of the brain, and the corpus striatum. In four cases which I have examined, the process was in the temporal lobes, the parietal lobule, the mid-brain, and the corpus striatum. If the process is a mild one, the hemorrhage and exudate are absorbed, and the injured brain tissue is gradually replaced by connective and neuroglia tissue. In this way small foci of sclerotic tissue are formed and the patient may afterward suffer from symptoms due to this condition. In the severer cases the softening becomes more extensive, larger hemorrhages occur, and in one case I have seen a massive apoplexy as the terminal stage.

Treatment.—The patient should be kept quietly in bed and should be given an active purge. Calomel is usually employed, but croton oil has seemed to me to be much more efficient as an eliminative and counter-irritant. The kidneys should be kept active, an ice cap placed upon the head, and leeches placed at the back of the neck. The treatment after this can be only that of sedation and support. If the patient is stuporous, and has a high fever, small doses of aconite should be given. If he is asthenic, he should receive strychnine. Of course, the nourishment should be carefully attended to, and, if he suffers pain, he should have morphine. Chloral and bromide seem to be the most efficient agents for relieving the restlessness and insomnia.

Multiple Sclerosis.

Multiple sclerosis is a chronic and progressive malady characterized by some paralysis, usually in the form of paraplegia, by coarse tremor, disturbances of speech, nystagmus, apoplectiform attacks, and various other cerebral and spinal symptoms depending upon the seat of the lesion. It is due to the development of sclerotic patches in the different parts of the brain and cord, which patches are for the most part the result of a neuroglia proliferation. The disease is probably an inflammatory rather than a degenerative one. It affects the spinal cord as well as the brain.

Etiology.—It occurs rather more frequently in the male sex and is a disease of the first half of life. Multiple sclerosis is in fact one of the few chronic nervous disorders of organic origin developing at this time. Most cases begin between the age of twenty and thirty. Cases have, however, been observed in infants and children, but the trouble in its typical form does not appear in the declining years of life. The sufferers have often inherited a feeble power of resistance on the part of the central nervous system. But the disease is not directly hereditary. There can be no doubt that the most important of all of the few causes of multiple sclerosis is infection. This is so true that it may be called a post-infectious disease. The infectious disorders which are followed by sclerosis are typhoid fever, pneumonia, malaria, and the eruptive fevers. Among these malaria and typhoid are the more important. It has been known to follow also diphtheria, whooping-cough, erysipelas, dysentery, cholera, and even rheumatism. Besides infection, trauma and shock are rarer causes, and a malady somewhat resembling multiple sclerosis may follow sunstroke. Syphilis is rarely a cause.

Symptoms.—The disease begins insidiously. A comparatively short time after recovery from malaria or some acute fever, the patient begins to suffer from weakness of the lower limbs with stiffness and some degree of numbness. The bladder is also a little weak, and it is difficult to retain the urine. In fine, the symptoms are very much like those of the onset of myelitis. Very soon the patient notices some unsteadiness in the gait, due not alone to weakness in the legs but to a certain degree of ataxia. He finds also that his hands are trembling and that this tremor increases upon voluntary motion. It is the type of tremor known as "intentional." He has at this time also some indistinctness in speech, it being difficult for him to enunciate long words. These come out in a slow, *syllabic* utterance, as it is called, each syllable being spoken separately. He may have also a little trouble in swallowing. By this

time he has had some sensation of numbness in the limbs, and some pains occasionally in the joints and extremities, but the sensory troubles are not very marked. If he is examined by a physician three or four months after the beginning of the malady, it will be found that the gait is stiff and awkward, the patient walking somewhat like a drunken man; or, in other cases, it may simply be the stiff, weak gait of moderate paraplegia. The Romberg symptom will be found to be present to some extent. The knee jerks are exaggerated and ankle clonus may be present. The hands are unsteady, and the movements are characterized by a jerky tremor, which may be so great that the patient has difficulty in dressing and feeding himself. This tremor disappears almost entirely if the patient lies flat upon his back. If he sits up, however, it may be seen perhaps in the muscles of the neck, causing the head to be oscillated, and constant, more or less regular tremor in the arms is present. The speech is thick and slow, and often almost unintelligible in the severer cases. Examination of the eyes shows a nystagmus, perhaps, only when the eyes are turned to one side, but often the jerky movements are seen, even when the patient is told to look directly at an object. The tongue is protruded in a jerky way, and attempts at swallowing are often awkward. If a glass of water is handed him, the patient seizes it, but in carrying it to his mouth he agitates it so violently that the fluid is spilled and perhaps the tumbler drops from his hands. Examination of the muscular system shows some weakness of the legs or possibly some degree of hemiplegia. There is, however, no marked atrophy of the limbs, and no change of any moment in the electrical reactions. Examination of the cutaneous sense may show some little tactile anæsthesia in the limbs, but this is not always present. There is a certain amount of ataxia, which is not so much due to the muscular anæsthesia as to inability to control and co-ordinate the movements. There is no loss of sense of weight, or of pressure. Of the nerves of special sense the eye is most frequently involved. The patient may have some diplopia from paralysis of one of the eye muscles. The pupils react to light and accommodation. Examination of the fundus of the eye shows a certain amount of atrophy of the optic disc in the temporal half, a characteristic condition in this disease. In later stages this may go on to complete and general atrophy of the nerve. On account of this involvement of the optic nerve, the patient suffers from contraction of the visual field, scintillating scotomata, and weakness of vision.

Sometimes he develops the above symptoms to only a moderate extent a short time after the infectious fever. He then gradually improves and gets nearly well. Some traces of the malady, how-

ever, still linger. After a number of years it begins to develop again, and it then progresses steadily. Thus an examination of the history of a case in which the disease apparently began well along in adult life will show that the beginning of the trouble dated back before the time of adolescence.

While the disease is running the course just described, the patient sometimes suffers from attacks of vertigo, and occasionally from sudden seizures resembling apoplexy, and even epileptiform attacks may occur. The mind is usually not much affected. There may be some slight dulness of the intellect, some hebetude, or even a slight amount of melancholia. In certain cases the patients are subject to attacks of impulsive laughing; that is to say, without any sufficient cause they suddenly break out in exaggerated laughter, from which they quickly recover themselves. These attacks may be frequently repeated.

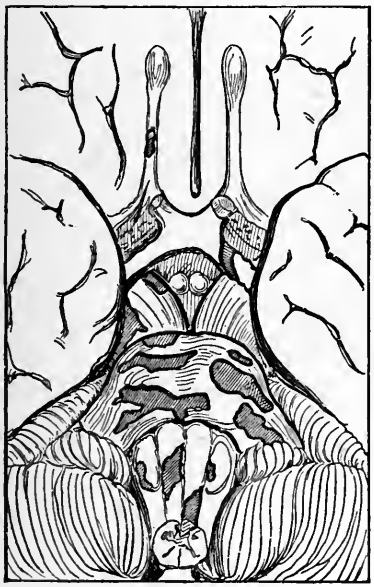
The progress of the affection in very variable. It sometimes goes steadily on without remissions, reaching finally in one or two years a chronic stage, in which the patient remains for several years without much change. At other times the progress of the disease is hastened by repeated exacerbations, accompanied by apoplectiform or hemiplegic attacks. In still other cases the amelioration continues and remains permanent, and a practical cure takes place. The various symptoms of the disease are classified as cerebral, cerebellar, and spinal. The cerebro-cerebellar symptoms consist in modifications of speech, attacks of vertigo and apoplectiform seizures, hemiplegia, intention tremor, mental changes, optic atrophy, and spasmodic laughing or crying. There is sometimes also a certain amount of deafness and perversion of taste and smell. Finally, lesions in the brain may give rise to a cerebellar ataxia, due to the development of the disease in the cerebellum.

Under the head of spinal symptoms are included the spasmodic paraplegia, with some bladder and sexual weakness, and a slight amount of sensory trouble. There may be also some weakness of the arms. Occasionally there are noted some trophic troubles, such as splitting of the nails and atrophy of the muscles.

Aborted Types or "Formes Frustes" of Multiple Sclerosis.—In some cases the nodules of sclerosis are so limited in number and so peculiarly placed that they give rise to very atypical forms of the disease. Perhaps the most common one is that in which the disease takes the type of a *progressive spastic paraplegia.* The patient suffers from weakness of the lower limbs, accompanied by stiffness, cramps, exaggerated reflexes, and disturbances in the bladder and rectal functions. Anæsthesias, pain, and the girdle

symptom may develop. In addition to this, however, a close examination will show some evidence of disease of the optic nerves and perhaps disturbances of the eye muscles. The patient will have nystagmus, diplopia, or other visual disorders. There will also be some attacks of vertigo or of epileptoid convulsions. The combination of the eye symptoms with the progressive paraplegia will often reveal the true character of the disease.

Pathology.—Grayish nodules are found distributed through the brain and spinal cord. They vary in size from a millimetre to two or



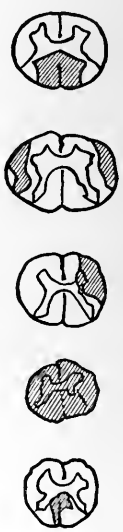


FIG. 205. FIG. 206.

FIGS. 205 AND 206.—MULTIPLE CEREBRO-SPINAL SCLEROSIS (Charcot).

three centimetres in diameter (one-twenty-fifth to one inch). They are of firmer consistence than is the surrounding brain substance, but are not quite so hard as is ordinary connective tissue. They consist microscopically of fibrous tissue which does not seem to be connected with the walls of the blood-vessels, as a rule. Very often the axis cylinders of nerves can be seen passing through the lesion. The nodules are found most frequently in the white matter of the brain, more especially in the pons, internal capsule, and centrum ovale (Figs. 205, 206). They rarely begin primarily in the gray matter, but may invade it secondarily. The roots of the peripheral, especially of the cranial, nerves occasionally contain or are surrounded by these sclerotic masses. In the spinal cord they may

extend up and down the gray and white matter for a considerable distance, or they may involve the whole cord at a certain level, turning it into a fibrous mass. The blood-vessels surrounding and in connection with these diseased areas show some evidences of thickening and increased vascularity, but no true inflammatory process. The primary pathological change in multiple sclerosis is as yet unknown; many things point to its starting originally from small emboli or thrombi which lead to minute softenings, with a secondary reparative and sclerotic process. The fact that the disease follows infective fevers makes such an origin of it seem probable. On the other hand, pathological anatomy does not yet bear out this view, and it is possible that the disease begins by a primary degeneration affecting first the myelin sheaths of the nerve fibres, this being followed

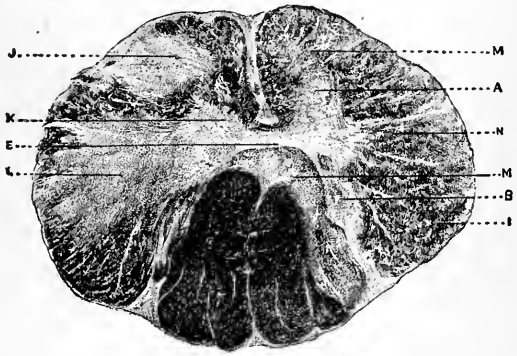


FIG. 207.—SPINAL SCLEROSIS. *J*, *K*, *L*, *M*, sclerotic foci; *A*, anterior, *B*, posterior horn (Blocq)

by a neuroglia and connective-tissue proliferation which ends in the formation of the small islands of sclerosis. An important pathological peculiarity of the process is that, while it destroys the myelin sheaths of the nerves, the axis cylinders remain intact for a long time, and consequently conduction of nerve impulses takes place imperfectly, directly through the nodular masses.

Course and Duration.—The disease runs a very irregular course. Its prodromal stage is long and remissions of considerable length occur. The disease may last from five to fifteen years, the average duration being five or six years. Death sometimes occurs from involvement of the nerves of the medulla, but more often from weakness and exhaustion or some intercurrent malady.

Diagnosis.—The diagnosis in typical cases is not very difficult; but as, on the other hand, typical cases are not common, the disease has always to be studied with great care before certainty can be reached. The diagnosis is based upon the slow development of

the disease, with attacks of vertigo, weakness, and uncertainty in gait; also upon the paralysis of the extremities with intention tremor, ataxia, rigidity, and contractures; upon the disturbances of vision, nystagmus, and the speech troubles. The presence of headache, attacks of vertigo, apoplectiform attacks, and the peculiar mental condition often furnish help. The age of the patient and the cause should also be taken into consideration. The disease must be distinguished from Friedreich's ataxia, spastic spinal paralysis, locomotor ataxia, dementia paralytica, bulbar paralysis, paralysis agitans, chronic meningitis, and hysteria. The points already given and those furnished under the heads of these different diseases must be utilized in making these distinctions. The method of exclusion may be used with advantage in reaching the diagnosis of this protean malady.

Prognosis.—The prognosis, while not favorable as regards the ultimate cure, is somewhat favorable as regards the remission and improvement, and the disease on the whole is not so severe as is locomotor ataxia or the other degenerative disorders.

Treatment.—In the treatment the same measures recommended for other chronic diseases of the nervous system must be employed. Hygienic measures, electricity, and hydrotherapy have some therapeutic value. Internally the use of large doses of iodide of potassium, the hypodermic injection of arsenic, the administration of nitrate of silver and of quinine and other tonics are advised. A very regular, systematic, and quiet mode of life, combined with the use of iodide of potassium and bichloride of mercury, has produced the best results in my experience, even in cases which gave no history of syphilitic infection.

THE APOPLEXIES.

Apoplexy is a clinical term used to indicate a condition characterized by sudden paralysis, usually attended with loss of consciousness, and due to the breaking or blocking up of a blood-vessel in the brain.

Apoplexy is a general term. Particular forms are described in accordance with the cause of the apoplexy. These are:

1. Intracranial hemorrhage, from rupture of a blood-vessel (Hemorrhagic apoplexy).

2. Acute cerebral softening, from embolism or thrombosis (Embolic or thrombotic apoplexy).

Apoplexy from Intracranial Hemorrhage (Cerebral Hemorrhage, Hemiplegia).

There are four groups of blood-vessels in the brain, those of the dura mater, those of the pia mater, and those supplying the basal ganglia and white matter. Besides this, we may consider the pons, medulla, and cerebellum, which are supplied chiefly by branches of the vertebrals as a separate group, subject to somewhat different mechanical conditions. Corresponding to this we have:

1. Dural or pachymeningeal hemorrhages.
2. Pial or subarachnoid hemorrhages.
3. *Central hemorrhages.*
4. Hemorrhages in the medulla, pons, and cerebellum.

It is the central hemorrhages (No. 3), due to rupture of the blood-vessels going to the great basal ganglia, internal capsule, and white matter, that constitute the great majority of cerebral hemorrhages seen by the physician. It is this class that I have particularly in mind in the following description.

Etiology.—At the time of birth and during infancy there is a slight tendency to intracranial hemorrhage owing to the accidents and injuries of labor. After this period the liability is very small, but slowly increases up to the age of forty, when predisposition specially begins. Four-fifths of all cases occur after forty, and the tendency to hemorrhages increases in each decade up to eighty, when it diminishes absolutely and relatively.* Males are slightly more predisposed than females (five to four). Rather more cases occur in cold weather, at high altitudes, in the temperate zone, and among civilized races. Heredity has an undoubted though not great influence in predisposing to cerebral arterial disease. Infective fevers and marasmic states are predisposing causes. Chronic kidney disease is present in one-third of the cases. Chronic alcoholism, syphilis, and gout are powerful predisposing causes. Rheumatism is less important. Heart disease, fatty and atheromatous arteries, arteritis, and miliary aneurisms may be regarded as more than simply predisposing—they

* Among 53 cases collected by me at Bellevue Hospital, the ages were: 10 to 20, 4; 21 to 30, 6; 31 to 40, 10; 41 to 50, 11; 51 to 60, 7; 61 to 70, 10; 71 to 80, 5. The right side was affected in 23 cases; the left in 25 cases. The location was: Pachymeningeal, 7; pial and cortical, 8; ventricular, 23; corpus striatum and vicinity, 7; optic thalamus, 2; corpora quadrigemina, 1; pons, 1; cerebellum, 3.

are determining causes. Leucocythæmia, scurvy, and purpura are conditions which also particularly tend to cause hemorrhage. The so-called apoplectic habit — short thick neck and high shoulders and florid face — has really some importance in the better classes. Congenital anomalies, such as a narrow thoracic aorta or inherited deficiency in the strength of the walls of the blood-vessels, also play a part. Any sudden physical exertion, such as straining at stool, the excitement at coitus or of a passion, eating a large meal and drinking a great deal of fluid, especially alcohol, taking a cold bath, all may lead to rupture of an artery.

The *symptoms* are the prodromal, those of the attack aud acute stage, and those of the chronic stage.

Prodromal symptoms are rare except in syphilitic cases. When present the patient suffers from dizziness, numbness of the hand and foot on one side, and a failure of memory for words. He may have "full" feelings or even pain in the head and bad dreams at night. Nosebleed and irregular heart action sometimes occur. The attack always comes on suddenly and may be accompanied (1) by convulsions and coma, (2) by coma alone, or (3) it may come without loss of consciousness.

1. Initial convulsions are rare and generally mean a meningeal hemorrhage. When present they are unilateral or partial, as a rule, but may be general. 2. The common mode of onset is with coma. The patient, without warning, suddenly becomes dizzy, loses consciousncss, and falls. The face is flushed, the pulse hard and rather slow, the breathing is labored and stertorous, the cheek on one side puffs out with each expiration, the eyes are partly closed, the eyeballs fixed or deviated to the paralyzed side, the pupils are contracted and rigid, the skin is bathed in sweat, the limbs are relaxed, but some evidence of hemiplegia is present; the urine may be retained or it and the fæces involuntarily evacuated. The urine is usually of rather high specific gravity and often contains albumin, even when there is no renal disease. The temperature in severe cases may fall below normal during the first twelve hours, even to 96° F., but this is not the rule. It is the rule, however, for the temperature in a few hours to be ½° or 1° higher on the paralyzed than on the sound side. If the case is rapidly fatal coma continues, respiration often assumes a Cheyne-Stokes character, the pulse becomes faster, the temperature gradually rises, and usually reaches 102° or 103° F., until just before death, when it may sink again. Swallowing and speech become difficult, hypostatic pneumonia sets in, and the patient

dies in from two to four days. In slower fatal cases the patient regains consciousness partially and then enters a condition of stupor or mild delirium. He is restless and suffers from headache. The temperature may continue normal for a time, but is usually higher on the affected side. At the end of two or three weeks it rises higher, pneumonia develops, the patient becomes unconscious, and death ensues. In the favorable cases, which constitute the majority, coma, if present, gradually passes away in from one to six hours, leaving the patient's mind somewhat weak and confused and his speech disturbed, or more rarely the intelligence may not be at all disturbed. During the first few days or weeks after the attack the physician finds that the prominent symptom is the hemiplegia. This affects the arm and leg most and the face least. Only the lower two branches of the facial nerve are involved, and the patient can shut the eyes. The tongue, if protruded, turns to the paralyzed side; the uvula is turned in various ways and its position is of no significance. There is often some evidence of cutaneous anæsthesia of the paralyzed side, and less often hemianopsia and disturbances of hearing occur. In right-sided hemiplegia the patient, after recovering consciousness, is often unable to talk or to understand what is said. Examination shows that he has a motor or sensory aphasia (*vide* Aphasia).

The deviation of the eyes and head to one side usually disappears in a day or two. Occasionally there is a temporary ptosis. The pupils at first are contracted, that on the paralyzed side the more so; this condition disappears with returning consciousness.

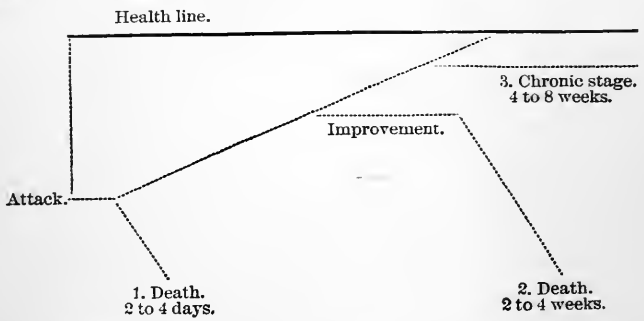
The paralysis of the arm and leg is flaccid at first, and the limb falls heavily when lifted; the reflexes are lessened or abolished.

Sometimes, however, rigidity sets in at once. This symptom occurs when blood has broken into the ventricles, and also in some meningeal hemorrhages. The skin reflexes are abolished or nearly so on the affected side.

The usual course of the temperature is for it to rise on the second and third day to 100° F. or 102° F., being $\frac{1}{2}$° to 1° higher on the paralyzed side. In a few days it gradually falls, so that by the eighth to the tenth day it is normal.

If the temperature continues to rise after the fourth or fifth day, it is a sign of an extension or inflammatory reaction of the hemorrhage. Hence the thermometer furnishes a very important criterion of the seriousness of the case.

The varying course of the apoplexy is shown in the following diagram:



The Chronic Stage, Hemiplegia.—At the end of a month, if fever and symptoms of cerebral irritation have subsided, the chronic stage may be said to begin. The hemiplegia has improved, the patient can move the leg and arm a little, sensory symptoms have lessened, the mind is clear, headache has disappeared. Improvement continues, though more slowly, for several months or even one or two years. During this time the patient is "a hemiplegic" (Fig. 208).

The hemiplegia affects the arm more than the leg, and the face least of all. The distal segments of the limbs, the feet and hands, are affected more than those near the trunk. The muscles that act bilaterally, such as those of respiration, phonation, and facial expression, are but slightly involved. The paralysis is not strictly a hemiplegia, for the muscles on the sound side are somewhat weakened, as tests will show. In severe cases, especially in old people, even the visceral muscles, especially those of the bladder, are weakened. At the onset of the attack there is sometimes a temporary "initial" rigidity of the muscles on the paralyzed side, or an "early" rigidity may develop in one or two days. There always develops at about the beginning of the second week a "late" rigidity. This, which at first is slight, gradually increases, and finally contractures affect the paralyzed limbs. The superficial reflexes, which at first were absent, reappear; the tendon reflexes become much exaggerated, and clonus can be obtained in the leg and arm. The sound side shares to a small extent in these conditions. The contractures affect the extensors of the foot more than the flexors, and bring the toe down and the heel up. The leg is held nearly extended, and the limb in walking is swung around, the toe scraping the ground. The shoulder is adducted, the forearm flexed, and the fingers are tightly shut into the palm by the

overaction of the flexors (Fig. 208). The facial muscles show a slight contraction and drawing to the affected side. The muscles on the paralyzed side do not waste. In infantile hemiplegia, however, the affected limbs grow less than those on the sound side.

FIG. 208.—A CASE OF CHRONIC HEMIPLEGIA WITH CONTRACTURES FROM CEREBRAL HEMORRHAGE (Curschmann).

The paralyzed limbs may be the seat of peculiar disorders of movement. These consist of

Associated movements.

Tremor.

Ataxia.

Choreic movements.

Continuous or athetoid movements (Fig. 209).

Spastic movements and cramps.

Such movements, aside from those that are spastic, are rarely seen in the hemiplegia of adults.

The electrical irritability may be at first slightly increased or diminished, but the change is small in amount and never reaches the degenerative stage.

Hemianæsthesia, if present at first, disappears to a great extent, leaving only residua about the feet and hands. Paræsthesiæ are common. In rare cases the patient suffers great pain in the arm and leg. This pain is generally of a burning character and very obstinate and distressing. Cramping pains in the legs and arms are common in the severer cases.

During the first few weeks after the onset joint inflammations and bedsores may attack the affected side. The temperature of the hemiplegic side is usually a very little higher than that of the sound side.

Vasomotor disturbances, sweating, skin eruptions, and increased growth of hair are some of the rarer symptoms.

The mental condition is more or less affected. The patient becomes irritable, cries easily, and is in general more emotional.

The memory is impaired, and the power of concentrating the attention and carrying on work is less. Sometimes a progressive mental deterioration sets in and epilepsy or insanity develops. The mental disturbance is greater in old people and depends somewhat on the size of the hemorrhage. Those forms which produce serious aphasia especially limit and lessen mental activity.

Meningeal Apoplexy.—Aside from the apoplexies due to rupture of the central arteries and involvement of the basal ganglia just described, there are a minor number in which the meningeal ar-

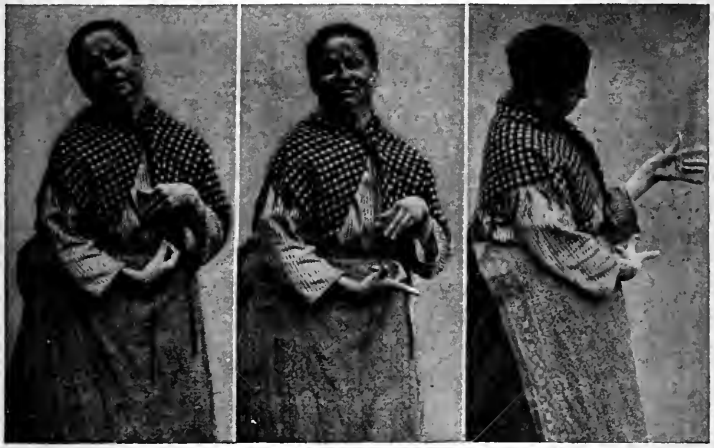


Fig. 209.—Showing Athetoid Movements of Hands (Curschmann).

teries, the cerebellar, or some branch of the basilar are affected; hence we have meningeal, cerebellar, and pons hemorrhages.

Hemorrhages from the vessels of the dura mater are usually due to a rupture of the middle meningeal artery or vein or some of its branches, and this is especially true in such hemorrhages as are the result of injuries to the head. The causes are injuries to the head, including obstetrical injuries, alcoholism, and insanity.

In dural hemorrhages the *result of head injuries*, the clot is sometimes intradural, lying in the arachnoid space, and sometimes epidural, lying between the bone and the dural membrane. The extradural hemorrhages are perhaps a little more common. In over one-half of these there is an interval of consciousness lasting from a few hours to two months, but usually only a few hours, between the accident and the time when distinctive cerebral symptoms develop. Then the patient gradually becomes dull, somnolent, and finally comatose. In about one-half the cases this interval of consciousness between the accident and the development of hemiplegia

is present. Along with the gradual or rather sudden loss of consciousness there develops a hemiplegia upon the side opposite the clot. This is usually not complete, though it may become so. Anæsthesia is rarely present. The reflexes are generally somewhat exaggerated, and there may be considerable rigidity. Spasmodic movements of some kind occur in nearly half the extradural cases and in more than half of the intradural. These spasmodic movements may involve the whole of the affected side, or may simply affect the eyes and the facial muscles. They consist of irregular twitchings. The pupils are usually somewhat contracted, more so upon the paralyzed side. When there is a dilated pupil on the side of the lesion and a small pupil on the opposite side, it is known as the *Hutchinson pupil*, and means a severe brain compression involving the third nerve at the base. The eyes are generally both turned toward the affected side and away from the lesion. The pulse is slow and full; the respiration is rarely stertorous, though it may sometimes be so, and Cheyne-Stokes respiration may be present. In these cases the clot is very large and the compression great. Aphasia may be present if the clot is upon the left side. The temperature may be raised one or two degrees, or it may be normal. The progress of the disease is usually steadily fatal unless surgical interference is undertaken. The coma deepens, the respiration becomes stertorous and then embarrassed, the pulse gets rapid and weak, and the patient dies. With surgical interference (since 1886), between two-thirds and three-fourths of the cases are saved (Scudder and Lund).

Dural hemorrhages *occurring idiopathically* are due sometimes to the rupture of a meningeal artery, and sometimes to rupture of the veins of the pia mater. This idiopathic hemorrhage is rare in ordinary practice, but is not specially so in insane asylums or in large city hospitals. This is because the two great causes of this type of hemorrhage are insanity and alcoholism. General paresis is the form of insanity with which it is oftenest associated. In the case of alcoholics, it is probable that injuries from blows are an exciting factor in the production of the hemorrhage, these occurring while the patient is in a state of intoxication. The symptoms of idiopathic hemorrhage are extremely variable, owing to the complicating influences of the insanity and alcohol. The patient after suffering from headaches or vertigo becomes suddenly comatose and shows marked evidences of hemiplegia and even of anæsthesia. Rigidity of the paralyzed side is often present, and sometimes spasmodic movements are observed. On the other hand, at times the paralysis can hardly be observed, and the patient is in a semicoma-

tose state, has a muttering delirium, and presents the general aspect of a person suffering from the œdema or "wet-brain" of alcoholics. In dural hemorrhages occurring in paresis, the patient usually without warning becomes unconscious, and he often has some convulsive symptoms and a hemiplegia develops. In these cases there is often a rapid improvement, and the patient gets partly well, usually experiencing other attacks later.

Pial apolexy occurs very rarely, and the most frequent cause is trauma associated perhaps with syphilis and alcoholism. In many instances very slight localizing symptoms occur, and no absolute diagnosis can be made. If the hemorrhage, however, is in the motor area of the cortex, local spasmodic movements and some hemiplegia are observed. The most characteristic symptoms are the sudden incomplete hemiplegia, involving, perhaps, mainly an arm or a leg, associated with local spasmodic movements, resembling Jacksonian epilepsy.

Pons Apoplexy.—This is accompanied by initial loss of consciousness and sometimes with spasmodic, jerking movements of the limbs, more particularly of the legs. Some rigidity on both sides of the body may be present. The facial or ocular nerves may be involved, and speech, articulation, and swallowing may be affected. The pupils are often contracted almost to a pin point, and the respiration is slow. The temperature almost always rises, and may reach as high as 103° or 104° F. There may be some disturbance in sensation and some hemiplegia. These hemorrhages are usually fatal.

Cerebellar Apoplexy.—Hemorrhage into the cerebellum occurs in one or two per cent of all fatal cases. Its recognition is very difficult. There is sometimes a preliminary period of severe headache, lasting several days. In other cases the patient at once falls into a state of profound coma, with stertorous respiration. Vomiting sometimes occurs. There may be some hemiplegia, and if so this is on the side of the lesion, owing to this pressure on the motor tract. Distinct evidences of hemiplegia, however, are not always observed. The condition of the pulse and arterial system is very much like that of ordinary apoplexy, but the respiratory system is usually more seriously affected. Disturbances in the movements of the eyes and in swallowing, and in fact all those symptoms which show a pressure or irritation due to blood oozing into the fourth ventricle may be present. Death is almost sure to occur, and is inevitable if the hemorrhage, as is so often the case, breaks through and reaches the fourth ventricle.

Pathology and Morbid Anatomy.—Spontaneous intracranial hem-

orrhage is always due to the presence of diseased blood-vessels in the brain. This diseased condition consists of:

1. A degenerative arteritis which results in producing small aneurisms. 2. A fatty degeneration of the vessel walls. 3. Besides this, in most cases the larger blood-vessels are atheromatous.

1. The arteritis produces small or miliary aneurisms which affect only the smaller arteries, especially those of the central group. They may be fusiform or sacculated in shape; they range in size from one-fourth to one millimetre ($\frac{1}{100}$ to $\frac{1}{25}$ in.) in diameter. They are usually not very numerous, but there may be as many as a hundred in the brain. They are the results, not of inflammation, but of a degeneration which affects first an area in the internal coat; this causes local weakness and consequent dilatation; secondarily there is a periarteritis. These aneurisms occur almost exclusively during the degenerative period of life.

2. Fatty degeneration of the walls of the small cerebral arteries occurs in purpura, scurvy, leucocythæmia, marasmic conditions, and post-infective states, especially in early life, and is the common cause of hemorrhage at that time.

3. Atheroma affects the larger vessels only. It is indirectly a cause of hemorrhage by lessening the elasticity of the vessel wall. Atheroma is present in from one-eighth to one-fifth of all cases. Hypertrophy of the heart is a factor in causing hemorrhage, and such hypertrophy exists in about forty per cent of cases. Emboli lodged in the cerebral arteries may cause hemorrhage by suddenly stopping the arterial circulation and raising the blood pressure. Hemorrhages are found by far the oftenest (twenty per cent) in the caudate and lenticular nuclei and adjacent parts. The lenticular and lenticulo-striate branches of the middle cerebral are oftenest affected; next the branches of the anterior cerebral to the caudate nucleus and the ventriculo-optic branches of the middle cerebral. The branches of the posterior cerebral break more rarely. The parts affected in hemorrhage, in order of frequency, are about as follows:

Caudate and lenticular nuclei.

Meninges and cortex.

Centrum ovale.

Optic thalamus.

Pons, cerebellum, medulla.

Cortex hemorrhages are generally small and may be subarachnoid or may break through into the arachnoid cavity. Ventricular hemorrhages are almost always secondary to a rupture into the neighborhood of the basal ganglia. Pons hemorrhages occur usually in the median line. Cerebellar hemorrhages are oftenest due

to rupture of the superior cerebellar artery. They usually cleave their way externally and break into the fourth ventricle. Dural hemorrhages are due to rupture of the meningeal veins and arteries and of the vessels in newly organized clots. They lie in the arachnoid cavity and flatten the convolutions.

The reparative changes after a hemorrhage take the following course: 1st, Coagulation of the blood, which in a few days begins

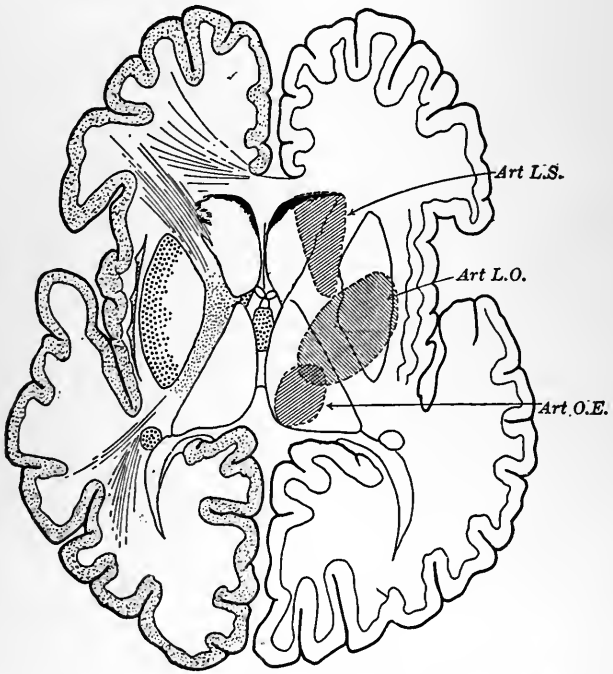


FIG. 210.—SHOWING THE DIFFERENT AREAS COMMONLY INVOLVED IN CEREBRAL HEMORRHAGE. *Art. L. S.*, lenticulo-striate artery; *Art. L. O.*, lenticular optic; *Art. O. E.*, external optic.

to soften and become absorbed. 2d. Formation of a fibrinous wall about the clot. This occurs from the seventh to the ninth day. 3d. Formation of a cyst with transparent fluid contents, and perhaps fibrous trabeculæ running through it, twentieth to thirtieth day. 4th. Contraction of the cyst wall, which begins by the fortieth day. 5th. Secondary degenerations begin from the tenth to the fourteenth day.

Physiology.—The blood pressure of the cerebral arteries is equal to about 155 mm. of mercury. The resistance or support furnished by the surrounding tissue is equal to about 10 mm. of mercury.

Hence there is a special liability to rupture of intracranial vessels. The middle cerebrals are most often affected, because they are in the most direct line from the heart and are nearest to that organ. The pressure lessens as the arteries subdivide and get farther away from the heart (Mendel).

The *diagnosis* of hemorrhagic apoplexy must be made from alcoholic coma, uræmic coma, opium coma, epilepsy and hysteria; acute softening from embolism and thrombosis.

From alcoholic coma the diagnosis is made by the odor of the breath, the incomplete coma, the equal pupils, the absence of low or unequal temperature.

From uræmic coma by the absence of albumin and casts in the urine, though their presence does not surely indicate uræmia; by the unequal pupils, the temperature, the absence of hemiplegia and of the physiognomy peculiar to cases of chronic Bright's disease.

From opium poisoning by the history, the stomach contents, the presence of equal and contracted pupils, the slow respirations, the temperature, and the absence of paralysis.

From epilepsy by the history of the onset with epileptic cry, the dilated and equal pupils, the biting of the tongue, the absence of hemiplegia, the rather rapid return of consciousness.

Hysterical attacks present little semblance to that of apoplexy; hysterical hemiplegia is characterized by its flaccidity, by its not involving the face, and by the presence of the anæsthesias and other hysterical stigmata.

In embolic softening the earlier age of the patient, the presence of decided valvular heart disease, the parturient condition, the slighter degree and shorter duration of coma, the absence of serious disturbance of temperature, the onset first of paralysis and then of convulsive movements and coma—all lead to a presumption in favor of embolism.

The presence, on the other hand, of a congested face, tense pulse, and throbbing carotids favors the existence of a hemorrhage.

From thrombotic softening diagnosis is more difficult. The occurrence of prodromata, consisting of slight seizures quickly recovered from, the slighter degree of coma, the advanced age, hard atheromatous arteries, evidence of anæmia and asthenia, weak or fatty heart, the absence of stertorous respiration, flushed face, and unequal temperature not much lowered or raised, the slight pupillary disturbance, and absence of convulsions point to thrombotic softening. Evidence of a lesion in the pons or cerebellum suggests hemorrhage, while evidence of lesion in the medulla points almost surely to softening.

The chances in any case between the ages of thirty and fifty, if there is no heart disease, are six to one in favor of hemorrhage.

Prognosis.—The majority of cases get over the first attack. They are very liable to have another within one to five years. The minority recover from this. Few survive a third attack. The prognosis of the attack itself depends on the severity of the coma and paralysis, the disturbance of temperature and of respiration, the evidence of rupture into the ventricles, the development of decubitus, the continuance of loss of control over the bowels and bladder.

If profound coma continues four days there is little hope; if fever develops and continues steadily, or if there is initial subnormal temperature, the prognosis is grave.

If the patient passes the first week with little or no fever and consciousness has returned, the prognosis is good.

The presence of renal disease and of alcoholism is bad. Development of slight delirium which continues is unfavorable.

Cerebellar and pons hemorrhages are very fatal, meningeal slightly less so.

The prognosis of the chronic stage has been given under symptoms.

Improvement continues rather rapidly for three months, then very slowly. Improvement may continue for one or two years. Complete recovery is very rare. The great danger after middle age is recurrence of the attack.

Treatment of the Attack.—The patient should be laid in a horizontal position and kept quiet. Ice should be applied to the head and hot bottles at the feet. The feet and legs should be swathed in cloths wrung out in hot water containing mustard, a cupful to a pail of water. A laxative should be given, either one or two drops of croton oil or a quarter of a grain of elaterium. If there is evidence of intense cerebral congestion, the pulse being very full and hard and the heart beating strongly, bleeding eight to ten ounces is justifiable. Ordinarily it is better to give a drop of tincture of aconite every twenty minutes for two or three hours. Pressure on the carotid of the sound side and even ligature of it have been recommended, but there is no experience yet to justify either. Administration of bromide of sodium and enemata of ergot have been advised, but are of doubtful value. After the first twelve hours, treatment must be symptomatic. Should delirium and other evidence of mental irritation appear, large blisters must be applied at the back of the neck and an elaterium purge given if the patient is not too weak. The use of iodide of potassium or mercury is not

indicated unless the case is distinctly syphilitic. The passage of a galvanic current through the brain cannot possibly do any good. Great care should now be taken that the patient does not develop pneumonia. The mouth and pharynx should be cleansed antiseptically, and the patient should not be allowed to remain in one position. If there is sufficient evidence of a meningeal or cortical clot, trephining should be seriously considered.

At the end of three or four weeks the faradic battery may be used carefully on the affected limbs. A séance of fifteen minutes daily for four to six weeks should be given, then treatment should be suspended for a fortnight, to be begun again and kept up systematically for a year if need be. Massage may be alternated with the electricity. When contractures develop the stabile galvanic current may be tried, though it does little good. Static sparks, however, are helpful; lukewarm baths should be tried and measures used to produce hyperextension of the affected parts.

Internally during this time the patient is to be given courses of iodide of potassium, tonics, and laxatives if needed. The patient should be made to live a quiet life, preferably in a warm, equable climate. The kidneys should be kept active and arterial tension low. For these purposes nitroglycerin should be given and at times small doses of chloral, and the diet should be simple and rather non-nitrogenous. Strychnine in very small doses (gr. $\frac{1}{100}$) sometimes helps the contractures; so also do the bromides and physostigma.

Acute Softening of the Brain (Embolism, Thrombosis).

Acute softening is a condition caused by the plugging of a blood-vessel with an embolus or thrombus, and is characterized by a sudden apoplectic seizure; the symptoms eventually running a course like those of cerebral hemorrhage.

Etiology.—Embolism occurs rather more often in women, thrombosis in men. Embolism is rare in children; it occurs oftenest between the ages of twenty and fifty, thrombosis betweeen the ages of fifty and seventy. The most important predisposing factors in embolism are acute or recurrent endocarditis, infectious fevers, profound anæmia, pregnancy, and blood dyscrasias; in thrombosis, syphilitic, lead, or gouty arteritis, fatty heart, and blood dyscrasias. The same causes which lead to the arterial disease which produces cerebral hemorrhage also predispose to thrombosis, though in the latter condition atheroma plays the important part.

Symptoms.—In embolism there are rarely any premonitory symptoms; the onset is sudden; it may begin with some convulsive twitchings, then follow hemiplegia and temporary loss of conscious-

ness. Coma, however, is rarer than in hemorrhage, and if present is usually shorter. There is rarely vomiting, nor do we find the hard, pulsating arteries, flushed face, and severely stertorous breathing. The initial temperature changes are slight, but in a few days fever may develop.

In thrombosis premonitory symptoms are frequent. In syphilitic cases there are headaches and cranial-nerve palsies. In other cases vertigo, temporary aphasia, transient hemiplegia, numbness of the hand and foot, and drowsiness may be present. The onset is more gradual; the hemiplegia slowly develops, taking several hours, perhaps, for its completion; meanwhile the patient gradually becomes comatose. The attack sometimes is rather sudden, with no loss of consciousness, and it may occur in sleep. The temperature often has a slight initial fall, followed by a rise, just as in nemorrnage. In both embolism and thrombosis the hemiplegia tends to improve very much in a few days or weeks unless the vessel obliterated is a large one. Embolism is rather more apt to affect the left side of the brain, though the difference is not great. The middle cerebrals are most frequently affected (seventeen out of twenty-seven cases). Softenings affect the vertebrals, basilar, and posterior cerebral arteries more often relatively than do hemorrhages; then the initial symptoms may not present the character of hemiplegia, but of a bulbar paralysis. Acute softening may kill within twenty-four hours, but, as a rule, the patient survives the onset, and if he dies it is not for several weeks. After the acute stage is over the patient passes into the chronic stage, which resembles in nearly all respects that of hemorrhage. After an acute softening, however, it is believed that there are more spastic symptoms and a greater tendency to mobile spasm. In embolism, owing to the youth and freedom from arterial disease, the mind is less affected; while in thrombosis the contrary is the case.

Pathology.—The embolic plug cuts off the blood supply from a certain area of brain tissue. In twenty-four hours this begins to soften. If the area is in the cortex it becomes red (red softening); if in the white and less vascular part, it is usually white with a few red punctate spots. The red softening gradually becomes yellow (yellow softening). The dead tissue softens and is absorbed, leaving a cicatrix or cyst. If the embolus contains infective microbes there may be a local encephalitis and abscess.

In thrombosis there are usually evidences of extensive atheroma or syphilitic arteritis. In those instances in which the thrombosis is caused by the blood state and a weak heart, little arterial change occurs. Atheroma affects chiefly the internal carotids and

the large arteries at the base, viz., the middle, anterior, and posterior cerebrals and the basilar and vertebrals. Thrombosis with apoplexy occurs oftenest in the corpora striata and optic thalamus, next in the pons and medulla. Embolism almost always affects the great basal ganglia or some cortical branch of the anterior and middle cerebrals. The secondary changes after thrombosis resemble those after embolism; a thrombus, however, may lead to supplementary embolism through breaking off of a clot, and both conditions may cause a complicating cerebral hemorrhage.

The Diagnosis.—The important points have been gone over under the head of hemorrhage. They may be tabulated in part here.

HEMORRHAGE.

Age, thirty to fifty.
Hereditary history of arterial disease.
Sudden onset, with coma and paralysis occurring together, the ıa deepening.
Initial and early rigidity.
Very unequal pupils.
Stertorous breathing and hard, rather slow pulse.
Peculiar alternating conjugate deviation.
Early rigidity.
Peculiar disturbances of temperature, as described.

ACUTE SOFTENING.

Earlier or later age.
History of syphilis.
Premonitory symptoms and more gradual onset (in thrombosis), more transitory coma or absence of coma.
Initial convulsive movements.
Presence of weak heart (in thrombosis) or endocarditis (in embolism).
Slight hemiplegia with anæsthesia.
The puerperal state (embolism).

Embolism is distinguished by the age, the presence of endocarditis, of the puerperium or infective fevers, and by the sudden onset, with perhaps some convulsive movements. Thrombosis occurs oftener in the aged, and there are prodromata—a slower onset and evidence of arterial disease and a weak heart.

The *prognosis* as regards the attack is somewhat better than in hemorhage, as a rule. In embolism it is good as regards recurrence; in thrombosis, bad. The mental condition is better in embolism; usually worse in thrombosis. The recovery from attacks is more

complete in acute softening. After the chronic stage is reached, however, the prognosis is about the same in all forms.

The *treatment* of the attacks consists essentially in rest and such attention to the bowels, kidneys, and heart as may be indicated. In thrombosis it may be important to give heart stimulants and arterial depressants, and for this purpose I advise the use of alcohol, digitalis, or strophanthus with nitroglycerin. Indide of potassium and mercury ought to be given if there is the slightest suspicion of syphilis. Later it is well to give courses of the iodides and mercury and of strophanthus, nitroglycerin, strychnine, and such tonics as may be indicated. The symptomatic treatment of the chronic stage is the same as in hemorrhage.

Cerebral Palsies of Children—Infantile Hemiplegia and Diplegia, Little's Disease.

The brain palsies of early life show themselves in the form of (1) hemiplegias; (2) diplegias or double hemiplegias, in which both sides of the body are involved; and (3) paraplegias, in which the lower limbs are chiefly or entirely involved. In these palsies, as in the same troubles of adult life, the loss of motor power is always accompanied by a rigidity and by some contractures and exaggeration of reflexes, in this respect distinguishing these paralyses from those of spinal origin. The seat of lesion in these cases is in the hemispheres of the brain, and it is the central motor neurons which are involved; that is to say, that part of the direct motor tract which extends from the brain cortex down to the spinal cord as far as the anterior horns. The brain palsies of children are therefore disorders of the cortico-spinal neurons, while the spinal palsies of children are disorders of the neuro-spinal neurons.

Etiology.—The disease occurs rather oftener in males than in females, though the difference is slight. The vast majority occur in the first three years of life; about one-third of them are congenital. Injuries to the mother during the time of pregnancy, possibly diseases and emotional disturbances at this time, are factors in producing the congenital cases. Those cases that occur at the time of birth are due to tedious labor, the use of forceps, and other injuries at the time of parturition. After birth, the causes are those which lead to the production of intracranial hemorrhages, embolism, and thrombosis; these being injuries and the infectious fevers. Of the latter, pneumonia, whooping-cough, measles, and scarlet fever are the most prominent. Syphilis is a rare cause; cerebro-spinal meningitis and epileptic convulsions are also occasional causes.

Symptomatology.—The disorder in about one-fourth of the cases

begins with a convulsion, which may be unilateral, but is usually general in character, and may last for several hours. At the same time a febrile process develops, and this continues for several days. When these acute symptoms have subsided, or before this, it is noticed that the child is paralyzed upon one side, the paralysis involving the arm, leg, and face, as in adult hemiplegia, or perhaps involving both sides. This paralysis undergoes gradual improvement, the face recovering earliest and most, the leg next, and the arm least. As the child develops it is found that the paralyzed side fails to grow as fast as the other, and there may be from one-half to one inch or two inches of shortening in the arm or leg. The circumference of the limbs is less, the surface somewhat colder, and some vasomotor disturbance may be present. With the progress of the case a rigidity of the affected limbs develops; the heel becomes drawn up, so that there is talipes equino-varus or equino-valgus. The flexors of the forearm and of the wrist and fingers contract, as do also the adductors of the thighs. In general it will be found that there is a contraction of the flexors and adductors of the affected limbs. With this rigidity and the contractures there are exaggeration of reflexes and clonus in most cases. In the disordered limbs the peculiar mobile spasms develop. These consist of athetoid, choreic, and ataxic movements, also sometimes tremors and associated movements. The choreic and athetoid movements are the most common (Fig. 209).

Along with the appearance of these symptoms it is noticed that there are disturbances in the mental condition of the child. It is usually backward in development, this backwardness ranging from simply feeble-mindedness to complete idiocy. Taking all cases, there is about an equal division between feeble-mindedness, imbecility, and idiocy (Sachs). Perhaps a little over one-fourth of the subjects have a fair intelligence. There is usually slowness in learning to talk, and in a small proportion of cases there is a decided aphasia. Such condition is rather more frequent with right hemiplegia than with left hemiplegia, though the rule is not an absolute one. In connection with the mental defect there may develop many of the peculiar moral traits associated with idiocy and low degrees of intelligence. Epilepsy very frequently complicates the disease; nearly one-half of the subjects suffer from this trouble. This epilepsy is in most cases general in character; in a few cases it takes the Jacksonian type, in a small number *petit mal* alone is noted. Examination of this class of sufferers reveals, aside from the paralysis described, various evidences of defective development. These are known as stigmata of degeneration; though they cannot be classed strictly among such, since they are acquired stigmata in

most cases, rather than marks which are the result of primary deficiency in development. These stigmata consist of a microcephalic or a macrocephalic skull, cranial and facial asymmetry, prognathism, imperfectly developed teeth, and a high palatal arch. It has been found that, as a rule, in cases of cerebral hemiplegias of childhood the patient eventually has a slight flattening of the skull on the side of the lesion (Fisher and Peterson). Finally, in a few cases there may be found defects in the special senses, such as imperfect hearing, deafness, deaf-mutism, and defects in vision such as hemianopsia, and perhaps imperfections in smell and taste. Anæsthesia is never observed.

The symptoms in the cerebral palsies of children, having passed the acute stage and having become somewhat ameliorated, enter into a chronic stage. This chronic stage begins within a few months after birth or after the onset of the disease. No great change occurs in the paralyses as the child grows older until he reaches the time of puberty, though there is a slight improvement in most cases. After the time of puberty, if the mental condition of the child is good, the physical symptoms are apt to improve considerably.

Morbid Anatomy.—The primary changes that lead to the cerebral palsies of children are: 1st, simple agenesis or lack of brain development, producing localized atrophy of the cerebrum and the condition known as *porencephalus* (true porencephalus is a condition in which, owing to a congenital defect in nutrition, a cavity or depression exists in the cerebral hemispheres, this cavity reaching generally into the lateral ventricle; true porencephalus is found in about one-fourth of the cases, though no definite statistics can be given, owing to the different interpretations given to this term); 2d, hemorrhage, which is probably the most frequent of the single causes; 3d, embolism; 4th, thrombosis; 5th, meningo-encephalitis and perhaps polio-encephalitis; 6th, a diffuse cortical sclerosis. Many other terms are used to describe the pathological conditions found at the basis of the brain palsies of children, but the principal causes of all are undoubtedly, as has been described, hemorrhages, embolism and thrombosis, and a defective development or agenesis. It is probable that in the hemiplegics the original lesion is generally a central hemorrhage, less often a meningeal hemorrhage. After this, probably the most frequent condition is a porencephalus from some intra-uterine accident, which may have been defective nutrition causing anæmia and softening, or hemorrhage, or thrombosis. Polio-encephalitis or inflammation of the cortex of the brain of the kind similar to polio-myelitis is alleged to be a cause in some cases by Strümpell, but this has not yet been proven. In

double hemiplegias or diplegias of children the cause is in the vast majority of cases a meningeal hemorrhage due to some injury or disturbance at the time of labor. In other cases of diplegia the lesion is a double porencephalus, which may be either the result of an intra-uterine hemorrhage or simply a defective development. In the paraplegias the lesion is probably very much the same as in the diplegias, that is to say, either a meningeal hemorrhage or a brain agenesis. Occasionally a diffuse sclerosis has been found in these

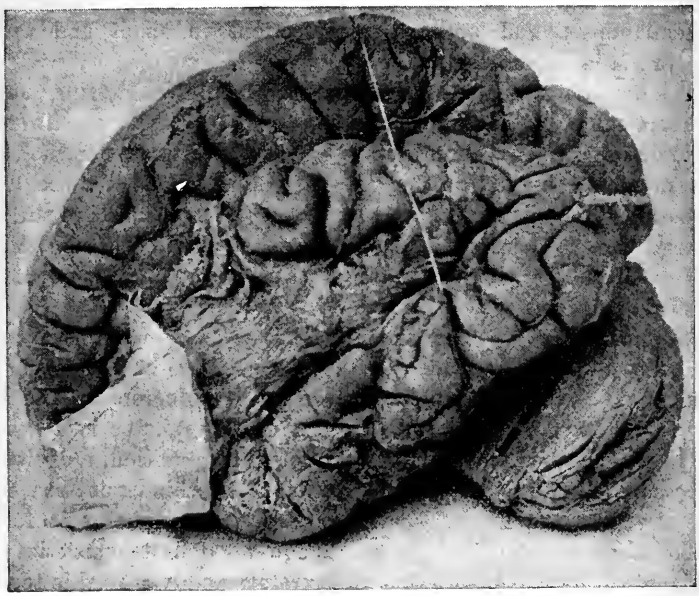


FIG. 211.—ATROPHIED BRAIN WITH SCLEROSIS AND A CYST, FROM A CASE OF INFANTILE CEREBRAL HEMIPLEGIA.

cases. Not infrequently, as the result of hemorrhages, there develop cysts which fill up the atrophied areas of the brain (Fig. 211). It is difficult to present accurately and definitely the relations between the pathological change and the clinical result, but it may be shown with some degree of correctness in the following table.

Original Lesions.	Later Pathological Condition.	Clinical Result.
Hemorrhage. Embolism. Thrombosis. Agenesis.	Atrophy. Lobar sclerosis. Cysts. Porencephaly. Microcephaly.	Hemiplegia. Diplegia. Paraplegia. Sensory defects. Mental defects. Epilepsy, etc.

Diplegia or Birth Palsies.—That form of the brain palsies of childhood characterized by double hemiplegias or diplegias has certain special characters which lead to its being often classed apart. These diplegias in almost all cases are congenital and are due either to injuries at the time of birth or to some disorders of intra-uterine life. There may be convulsions or a prolonged state of asphyxia at

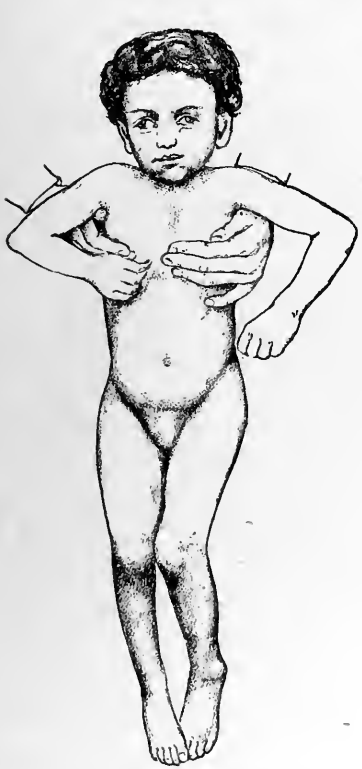


Fig. 212.

Fig. 213.

Figs. 212 and 213.—Cerebral Diplegia with Contractures and Talipes. Fig. 209, standing; Fig. 210, suspended by arms.

the time of birth. After recovery from this no special trouble is noticed with the child by the mother for some weeks or months, when it will be found that it does not use its arms or legs. Other convulsions develop, and eventually the features of a double hemiplegia with mental impairment and epilepsy are observed (Figs. 212, 213). In these cases the mental defect is much more decided than in the hemiplegias; indeed, few of these cases ever show any good amount of intelligence. Epilepsy is extremely common. The anatomical lesion in the cases is, as already stated, either a meningeal hemorrhage which has pressed upon and injured the cortical

motor areas in each hemisphere, or it is a congenital porencephalic defect.

Spastic Cerebral Paraplegia, Little's Disease.—In a few cases the brain lesion is such that there results little disturbance to the arms or face; the paralysis is largely confined to the lower extremities. The mental condition is often very good, and there is no epilepsy. As the child gets older great improvement in the use of the limbs may occur. There are, however, rigidity and contractures of the limbs, exaggerated reflexes and spasms of the flexors and the adductors, so that the child's legs cross each other and interfere in its feeble attempts at walking. The disease is thought to be due to a developmental defect in the motor tracts, and it is possible that in some cases only the spinal cord is affected.

Diagnosis.—The clinical diagnosis of cerebral palsies is to be made from the spinal palsies. The latter are distinguished by the fact that in the paralyses of spinal origin there is no rigidity or exaggeration of reflexes, and there are electrical degenerative reactions of the muscles and decided wasting of the limbs with shortening. The mode of onset in cerebral palsies and their distribution in the form of hemiplegias in which the face is involved also indicate the seat of the lesion. The pathological diagnosis is by no means an easy one. Cerebral palsies occurring at the time of birth and accompanied at that time by general convulsions or asphyxia may be considered to be due to meningeal hemorrhage, especially if the delivery of the child has been brought about by the use of forceps or if the labor has been long and tedious. Diplegias and paraplegias which are congenital are probably due to true porencephalus, provided there was no difficulty at the time of labor and there were no convulsions or other serious phenomena after it. Cerebral palsies occurring after birth in the first, second, or third year of life are apt to be due to hemorrhage, and less often to embolism or thrombosis. Hemiplegias developing after infectious fevers are likely to be due to hemorrhage. In diagnosticating the pathological lesion in such cases it must always be remembered that hemorrhage is much more frequent than embolism, and that thrombosis as a factor has not yet been very clearly established.

Course and Prognosis.—In all types of the disease the course is chronic and perfect cure is hardly possible, although in the slighter forms of hemiplegia nearly all traces of the paralysis may be absent. In the hemiplegic form the patient often reaches adult life, and if his intelligence is not defective and he has no epilepsy the motor trouble improves a great deal and he may live a long and useful life. If epilepsy and mental defect are present, there ensues eventually a further mental deterioration, and such subjects rarely

live much beyond the period of adolescence, or if they do they pass into the asylums for the idiotic and epileptic. The diplegic and paraplegic cases have a much worse prognosis both as to duration of life and as to improvement in symptoms, except occasionally the type described above as cerebral spastic paraplegia or Little's disease. The degree of intelligence and the absence of epilepsy are the two factors which measure the seriousness of these cases, as they do those of the hemiplegias. As regards the significance of individual symptoms, the post-hemiplegic movements have a bad import; the presence of a microcephalic head or of decided marks of degeneration is unfavorable.

Treatment.—The treatment, so far as the paralysis is concerned, is largely mechanical. The patient is benefited by occasional courses of electrical treatment which stimulate somewhat the nutrition and functions of the muscles. Massage and stretching of the contractured tendons and limbs also are helpful in my experience. The orthopædic surgeon is able to render valuable assistance by occasional overstretching the contractured limbs and placing them in splints. Tenotomy may also be resorted to with advantage, as I have had occasion to see. The child should be encouraged above all, however, to use the limb as much as possible. He should be taught gymnastic exercises; running, walking, and bicycle riding are all measures which give great help. When the child's intelligence is good and there is little or no epilepsy, a great deal can be expected in the way of improvement as the child grows older.

So far as the epilepsy is concerned, it should be treated on the same principles as idiopathic epilepsy, except that great care should be had in the use of the bromides; a thorough test must be made in order to determine how much of this drug will suppress the fits, and then its use must be graduated in the future in accordance with the knowledge thus obtained. The mental defects of the child can be helped only by proper training of the body and careful education of the mind. The question of operative interference in these cases has of late excited much attention. *A priori* it would not seem as though surgical interference could do good in relieving conditions in which there is destroyed or atrophic tissue. Still the subject must be dealt with empirically, and there have been some results which show that apparently a relief is obtained in a few cases by trephining the skull or by Lannelongue's operation of craniectomy. If there is no microcephalus, if the case is one of hemiplegia with imbecility and epilepsy, the surgeon should simply make an exploratory opening. If he then finds any evidences of compression from the presence of a cyst, this may be very cautiously opened.

If there is microcephalus, the linear craniectomy is the operation which is indicated. In all cases, in operating on children it has been found that it is imperative that the operation be made as short as possible, and that as little be done at any single operation as is consistent with the indications.

CHAPTER XX.

TUMORS OF THE BRAIN—SYPHILIS.

The kinds of tumor found in the brain are tubercle, syphiloma, glioma, and sarcoma, which are the common forms; myxoma, carcinoma, fibroma, osteoma, cholesteatoma, lipoma, psammoma, neuroma; vascular tumors including aneurisms; echinococcus, and cysticercus. In fact, all forms of new growths are found in the brain; but the infectious granulomata, tubercle and gumma, and the sarcomatous type of tumors are the most common. As compared with other organic diseases of the central nervous system, brain tumors are rare.

Etiology.—Brain tumors affect males oftener than females, the ratio being about as two to one (644 : 320). Sarcomata alone seem to affect females about as often as males. Brain tumors occur with about equal frequency throughout all ages of life up to about fifty; one-third occur under the age of twenty (Gowers). During childhood tumors are about equally distributed throughout all ages (Starr). One-half of all the tumors of childhood are tuberculous; after this come gliomata and sarcomata. The gumma, glioma, and sarcoma begin to be more frequent after the age of twenty. Sarcoma and especially cancer occur in the middle and later ages of life; but brain tumors of any kind are extremely rare after the age of sixty.

To sum up in tabular form, the relative frequency of the different kinds of tumors with regard to age is shown in the following:

Childhood, ...	tubercle, parasites.
Early life, ...	gumma, glioma, parasites.
Early and middle life, . .	sarcoma, glioma, and gumma.
Middle and late life, . . .	sarcoma, gumma, cancer.

Heredity has a slight influence in predisposing to brain tumors. Blows on the head and other forms of injury to the cranium are exciting causes in a small proportion of cases.

Symptoms.—The symptoms of brain tumors vary extremely in accordance with the location, the kind of tumor, the rapidity of growth, and the age of the patient. The general course of a case of brain tumor in an adult is somewhat as follows: The patient first notices a headache which is intense and persistent, and

which has exacerbations of frightful severity. With the headache or between the attacks vomiting occurs, which is often not accompanied by any nausea. Sensations of vertigo, annoying paræsthesias, and convulsive movements affecting one or more extremities develop, and there may even be general convulsions. The patient finds that his eyesight is weak and progressively deteriorates. The mind becomes more or less disturbed, the mental processes are dull and slow, a feeling of hebetude and incapacity to attempt any mental exertion is present. As the disease progresses the intense pains and vomiting produce weakness and emaciation. Paralyses of various kinds develop. Blindness may ensue. Convulsions of a local or general character become more frequent, and finally the patient becomes bedridden and helpless.

The course of the disease is not a steady one, there being often slight remissions, or there may be periods when progress seems to be arrested. After a period of time varying from one to four or five years death occurs from exhaustion or some intercurrent malady.

The symptoms thus very briefly outlined are divided into general and focal. The *general symptoms* are:

Headache,
Vertigo,
Vomiting,
Optic neuritis,
Mental defects.

Besides these there may be general convulsions and speech disturbances.

Headache occurs in from one-half to two-thirds of the cases; it is very severe and the pains are of a boring or lancinating character; they are so horrible that they often lead the patient to think of suicide. The pains are sometimes periodical, occurring every night or every other day, and suggest by their perodicity a malarial character. They are located sometimes in the brow or in the occiput, while sometimes they are diffused all over the head; they are rather more frequent than otherwise in the neighborhood of the tumor. They are more frequent with cerebellar tumors than with those located anywhere else. Pains are also frequent with tumors of the mid-brain and of the cerebral hemispheres. They are less frequent with tumors situated in the peduncles and at the base of the brain. The pains are due to the increased intracranial pressure and to irritation of the membranes of the brain by the encroachment upon them of the new growth. Headache occurs in about the same proportion in children and adults, and it does not seem to bear much relation to the kind of tumor, although the pains are generally less with the

gliomata, and they are more frequent with rapidly growing tumors whatever their character. With the pains there is often a local tenderness of the scalp and cranium which may be elicited by percussion, and in most cases there is greater tenderness in that part of the cranium lying over the tumor.

Vomiting is a symptom which is almost as frequent as headache. The vomiting is often of a projectile character and not accompanied by much nausea. Vomiting occurs, as does headache, more frequently with cerebellar tumors. It is associated with rapidly growing tumors such as syphilitic or tuberculous neoplasms.

Vertigo is a general symptom which occurs in from one-third to one-half of the cases. The vertigo may be slight, such as is often felt from ordinary causes. Occasionally it is very severe and accompanied by forced movements. The severer forms and those associated with forced movements occur with tumors of the cerebellum and the parts closely connected with it.

Optic neuritis is one of the most frequent and important of all the general symptoms of brain tumor; it occurs at some period of the disease in at least four-fifths of the cases, more frequently in cerebellar tumors and in those of the mid-brain and great basal ganglia. It is rare in tumors of the medulla. It is less frequent and marked in the slow-growing tumors. The neuritis may run a somewhat rapid course and then improve a great deal or even for a time disappear; but ordinarily the course is progressive and it ends eventually in an atrophy of the optic nerve. Hence the examination of the eyes in brain tumors should be made a number of times in order to note the progress of the trouble. Primary atrophy of the optic nerve does not occur in brain tumors. The inflammation almost always affects both nerves, but it may begin with one and subsequently affect the other.

Mental defects are almost always present in tumors of the brain. These defects consist in a slowness of the mental processes, a condition of hebetude, a tendency to attacks of somnolence, and sometimes a peculiar childishness and silliness or peculiar mental irritability. The memory is also usually somewhat weakened and the power of attention lessened. Such psychical defects are more frequent with tumors of the frontal lobes and more frequent also with large tumors.

General convulsions occur in about one-fourth of the cases and more frequently when the tumors are situated in the cerebral hemispheres and cortex. There may be also apoplectiform attacks, from which the patient recovers in the course of a few days or weeks. More rarely there is a genuine apoplexy from the bursting of a blood-vessel in the neighborhood of the tumor.

The speech disturbances are most marked in tumors which affect the pons and medulla and the origin of the cranial nerves. Such speech disturbances, when characteristic, are shown by a confluent articulation; that is to say, the patient runs the syllables together.

The cranial temperature in brain tumors is in most cases somewhat raised as compared with the normal (Gray, Mills, and Lloyd). The elevation may be several degrees above the normal. The normal average scalp temperatures (Gray) are from 92° to 94.5° F., being somewhat higher over the frontal and parietal than over the occipital regions. In brain tumors the temperature has been found raised to 95°, 96°, and 98°. The value of thermometric observations, however, in the symptomatology of brain tumors is somewhat doubtful, owing to the variability in the normal temperature and the difficulty of getting accurate records.

Focal Symptoms.—Having by a study of general symptoms arrived at a fairly certain diagnosis as to the presence of a tumor, it is necessary to corroborate the diagnosis and to localize the lesion by an examination of the symptoms which are the result of irritation or destruction of certain particular parts of the brain; these are called the focal symptoms. For purposes of special or local diagnosis we divide the brain into the following parts or areas (Knapp): 1. The prefrontal, which includes all that part lying in front of a line that extends from the upper end of the ascending branch of the fissure of Sylvius directly up at right angles to a horizontal line between the frontal and occipital poles of the brain (see Fig. 211). This region includes probably centres for the movement of the head and eyes, but it is chiefly concerned with the higher intellectual processes; its under surface lies on the orbital plate of the frontal bone and upon the right olfactory lobes. 2. The central region, which is bounded in front by the vertical line just described, behind by a line passing down from the anterior end of the parietal fissure to the fissure of Sylvius, and above by a line that bounds posteriorly the postcentral convolution. 3. The parietal lobe. 4. The occipital lobe. 5. The temporal or temporo-sphenoidal lobe. 6. The corpus callosum. 7. The great basal ganglia and capsules. 8. The corpora quadrigemina, deep marrow, and pineal gland. 9. The crura cerebri. 10. The pons and medulla. 11. The cerebellum. 12. The basal surface of the brain. The boundaries of most of these areas are indicated better by the figure than by a description. They correspond to some extent with the cerebral lobes, but not entirely so, since the frontal and parietal lobes divide between them the central area.

1. Tumors of the prefrontal area. Tumors in this area often

show no particular localizing symptoms, and this part of the brain is consequently put down as a latent one; nevertheless, in a good proportion of cases tumors here produce peculiar mental disturbances that, taken in conjunction with the general symptoms, enable us to make a local diagnosis. The symptoms are peculiar mental hebetude, childishness, irritability, often a kind of silliness and emotional weakness, a tendency to laugh and cry and to get angry at trifling causes. The entire character and temperament of the man are sometimes changed. Besides this, owing to implication of

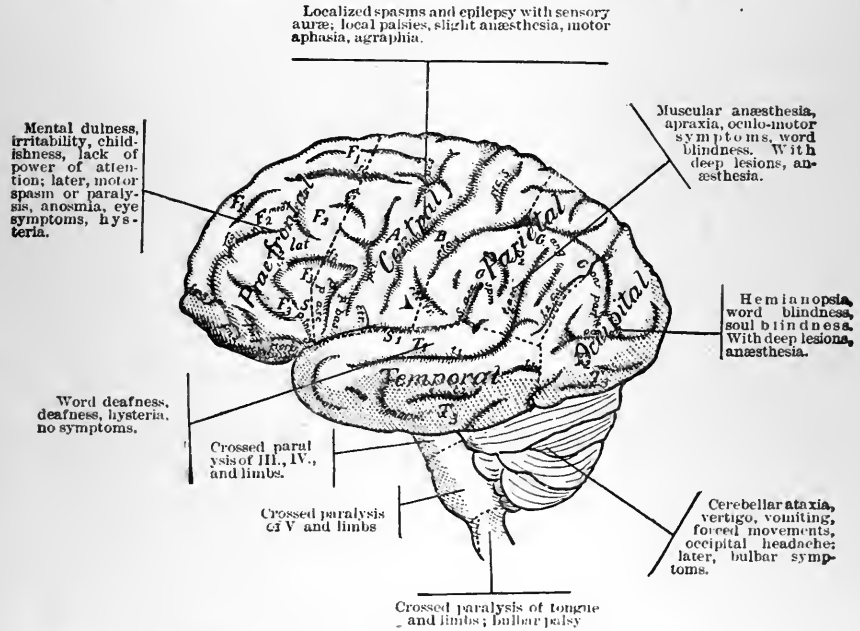


FIG. 214.—SHOWING THE FOCAL SYMPTOMS OF BRAIN TUMORS.

the olfactory nerve, there may be loss of the sense of smell on one or both sides; implication of the optic nerves will cause hemianopsia and optic neuritis. If the tumor involves the orbit there will be paralyses of the ocular muscles and protrusion of the globe of the eye. If the tumor grows backward there is gradual invasion of motor centres with irritation, showing itself by spasms, convulsions, and later by paralyses.

2. Tumors of the central region. It is in this area that we are often able to make the closest and most accurate diagnosis of the localization of new growths, owing to their involvement of the different motor centres. Through this involvement these centres are at first irritated, with the result of producing local spasms or Jackso-

nian epilepsy. Such spasms are often preceded by sensory symptoms or auræ. As the tumor grows the area of involvement becomes larger, spasms become more diffused, and general convulsions may finally appear, with hemiplegia. The motor disturbances are not infrequently accompanied by sensory disorders. These may be simply feelings of numbness or prickling, which either are permanent or simply precede spasms, or there may be hemianæsthesia of a moderate degree to pain, touch, and temperature. The muscular sense also may be somewhat involved. In cases of slight sensory involvement the capacity for localizing sensations seems to be most implicated. Besides the symptoms mentioned, there may also be motor aphasia and agraphia. The exact localization must be worked out with the help of the figures and descriptions given under anatomy.

3. Tumors of the parietal area. The symptoms produced by tumors in this area may be very slight. The most characteristic are disturbances of muscular sense, which occur when the supramarginal gyrus is affected, and word blindness, which occurs when the angular gyrus and inferior lobule are affected. When the tumor is higher up near the longitudinal fissure, the muscles of the lower limbs may be involved, and if the tumor encroaches upon the central area spasms and paralyses of various muscular groups ensue. The cortical representation of the third nerve is thought to be in the neighborhood of the angular gyrus, and some cases have been reported in which paralysis of this nerve resulted from tumors in that area.

4. Occipital lobes. Tumors in this region, if situated in the cuneus and first occipital convolution, produce homonymous hemianopsia. If the tumor involves the other parts of the occipital lobe and the cuneus is not seriously involved, there may be a condition known as soul blindness or incapacity to understand the nature of the things which one sees. If the tumor extends up chiefly toward the angular gyrus, there may be word blindness, along with some hemianopsia. If the tumor extends farther forward into the parietal lobe, there may be hemianæsthesia, hemiataxia, and perhaps a little hemiplegia owing to involvement of the fibres of the internal capsule.

5. Temporal area. The temporal or temporo-sphenoidal area on the right side is very nearly a latent one. On the left side tumors involving the posterior part of the first and upper posterior part of the second temporal convolution produce word deafness. Tumors in either lobe when large and extending well down toward the base may produce attacks of vertigo or forced movements, owing

probably to irritation of the internal ear. Tumors that involve the hippocampal convolution and the uncus may produce perhaps some disturbances in the senses of smell and taste.

6. Tumors of the corpus callosum. Tumors situated in this area are very rare. Their symptoms have been thought to be somewhat characteristic; but in the writer's experience they correspond closely with tumors situated in the third ventricle and lateral ventricles of the brain; in other words, tumors which, beginning in the central parts of the brain, gradually extend outward toward the periphery. The symptoms credited to tumors of the corpus callosum are, first, the general sympoms of brain tumor, to which there are superadded a gradually developing hemiplegia with later a paraplegia. At the same time there is a great deal of mental dulness, stupidity, and drowsiness; the patient often sits for hours mute, refusing to speak, or lies in a half-somnolent condition. There are no paralyses of the oculomotor nerves or of the other cranial nerves. There is no anæsthesia. The disease gradually progresses and the patient dies in coma.

7. Tumors of the great basal ganglia and the capsule (the opticostriate region). The general symptoms of tumors of this region resemble in many respects those of tumors of the corpus callosum. The stupidity, however, may be less marked. There is usually a progressive hemiplegia which may be accompanied by anæsthesia and sometimes by choreic movements, if the tumor involves the optic thalamus and adjacent part of the capsule. Tumors of the caudate nucleus alone and of the lenticular nucleus alone seem to give rise to no special symptoms, and these regions are regarded as latent. Tumors of the anterior three-fourths of the optic thalamus alone may cause no special symptoms, but in some cases there occur peculiar choreic or athetoid movements. These, however, are probably due to irritation of the fibres of the internal capsule. If the tumor involves the posterior part of the optic thalamus and adjacent areas, there will be a hemianopsia, which may be distinguished from the hemianopsia due to lesions in the occipital lobe by the presence of the *hemiopic pupillary reaction;* that is to say, a ray of light thrown in upon the insensitive part of the retina will not produce a reflex contraction of the pupil.

8. Tumors of the corpora quadrigemina, deep marrow, and pineal gland. The characteristic symptoms, as shown by Nothnagel, of tumors of this region are inco-ordination, forced movements, and oculo-motor palsies. Together with these there may be hemianopsia or blindness due to destruction of the primary optic centres. It is possible that some degreee of deafness or hemideaf-

ness may be produced by the involvement of the posterior tubercles of the corpora quadrigemina.

9. Tumors of the crus. Tumors of the crura cerebri are extremely rare. When present, they cause hemiplegia and perhaps a hemianæsthesia, with paralysis of the third nerve upon the same side as the lesion; in other words, a crossed paralysis (Fig. 215, *M*).

10. Tumors of the pons and medulla. Tumors in this area nec-

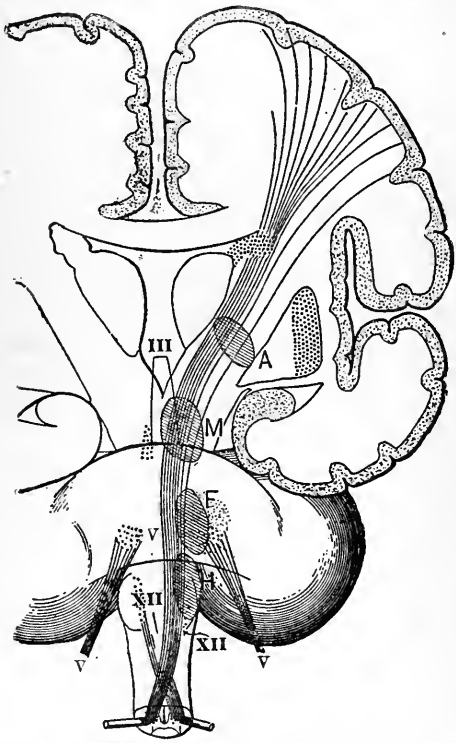


FIG. 215.—SHOWING THE MECHANISM OF CROSSED PARALYSIS. Lesion at *M* causes paralysis of third nerve, lesion at *F* paralysis of fifth nerve with hemiplegia of opposite side. (After Van Gehuchten.)

essarily produce very varying symptoms in accordance with their size and location. If the tumor is in the pons it will cause, if situated high up, a palsy of the third nerve on one side and hemiplegia on the opposite side. If lower down there may be a palsy of the fifth nerve on one side and hemiplegia on the other side (Fig. 215, *F*). If the tumor is extensive it may produce not only a hemiplegia, but a hemianæsthesia. If situated somewhat superficially and on the lateral edge of the pons involving the peduncles, there will be forced movements of the body, either toward or from the seat of the lesion.

If the tumor is in the medulla it will produce hemiplegia and hemianæsthesia, with paralysis of the hypoglossal nerve or perhaps some other cranial nerves upon the same side (Fig. 215, *II*). If large and involving both sides of the medulla, there may be the general symptoms of a progressive bulbar paralysis. One peculiarity of tumors situated in the pons is that they sometimes produce a conjugate deviation of the eyes which is away from the side of the lesion. In this respect the symptoms differ from conjugate deviation produced by lesion in the cerebral hemispheres, in which the head and eyes are turned toward the side of the lesion.

11. The general symptoms of tumors of the cerebellum are, as we have already said, more pronounced than those of tumors of other regions; we more frequently have headache, vomiting, vertigo, and optic neuritis from neoplasms here. If the tumor is situated in the lateral lobes of the cerebellum, no localizing symptoms develop until the tumor becomes very large so that it presses upon the medulla or other adjacent regions. When the tumor is in the middle lobe a peculiar ataxia develops, known as cerebellar ataxia. The gait of the patient is a reeling one like that of a drunken man, or in walking he takes short steps and spreads his legs as if in fear of falling. This has been called the titubating gait. Besides this, severe forced movements may occur which usually throw him sideways or perhaps forward, very rarely backward. Secondary symptoms from pressure on the medulla often develop in tumors of the middle lobe, such symptoms being glycosuria and disturbance of the functions of the cranial nerves. Late in the disease hemiplegia and paraplegia and bulbar symptoms may develop from extreme pressure. There may be also hydrocephalus due to pressure on the veins of Galen and obstruction of the return flow of blood from the central arteries of the brain.

12. Tumors of the base of the brain. Tumors situated in the anterior fossa produce symptoms very much like those described under the head of tumors of the prefrontal area, but there is necessarily destruction of the olfactory lobe and there is more apt to be involvement of the optic and oculo-motor nerves and of the tissues of the orbit.

Tumors of the middle fossa. Tumors sometimes involve the hypophysis. Such condition has been found in cases of acromegaly; but on the other hand a number of tumors of this region have been described in which none of the symptoms of acromegaly were present. Tumors of this region and of the interpeduncular space produce symptoms such as would naturally result from pressure on the optic chiasm, and it is mainly the early presence of optic neuritis

and of peculiar forms of hemianopsia which differentiates lesions in this area from those in the anterior fossa.

Multiple tumors. About one-seventh of all brain tumors are multiple. Hence in making a diagnosis of the localization of tumors this fact must be borne in mind. The tumors which are most frequently multiple are tubercle, cancer, and melanotic growths.

Pathology.—Tubercle is the form of tumor found oftenest in children and is altogether the most frequent of brain tumors. It is more often located in the cerebellum, but may appear in the pons or other parts of the brain. It may be a single or, as it is then called, a solitary tubercle, or there may be a multiple growth. The tumor is irregularly round in shape and varies in diameter from one and a half to two inches. It has a grayish-yellow appearance externally; internally, a yellowish or cheesy look. It is not vascular, but is often surrounded by softened or inflamed tissue. There may be an associated meningitis. The tumors, when solitary, usually start from the central parts of the brain, but they also develop on the meninges of the convexity, particularly in the parietal region, and sometimes they develop also at the base. Tubercle always arises from infection by the tubercle bacilli, which are carried by the blood to the brain. The tumors develop usually from some infectious focus, starting in a blood-vessel of the pia mater. Microscopically the tumor shows the ordinary appearances of tuberculous growths. It contains in its periphery many round cells, nuclei, and giant cells. In the centre there is usually an amorphous substance, the product of degeneration and the breaking down of the ordinary substance of the tumor. The characteristics of the growth are the presence of the round cells and giant cells, the caseation and softening of the centre, and the absence of vascularization, with the presence of the bacilli.

Syphiloma or gumma. Gummatous tumors of the brain are usually associated with syphilitic meningitis or some other form of cerebral syphilis, such as endarteritis, and perhaps inflammation of the cranial nerve roots. Syphilitic growths are usually found upon the brain surface, oftenest on the base, next upon the convexity of the frontal and central convolutions. The process appears either in the form of a somewhat distinct tumor or in the form of an irregular thickened exudate lying upon the surface of the brain and forming what is called gummy meningitis. The gummata may attain great size. They start usually from the pia mater and are due, as in the case of tubercle, to the irritative action of some infective organism. The gumma is irregular in shape; it has a somewhat thick grayish periphery and often a yellowish centre, the appear-

ances differing with the age of the tumor. Microscopically it is found to consist of small round cells and spindle cells with various broken-down nerve-tissue elements. It presents in the interior the evidence of cheesy degeneration, somewhat like that in tubercle, but less marked. There is a peculiar development of fibrous tissue in the syphilitic growths which distinguishes them somewhat. Besides this, the blood-vessels are numerous in the periphery and show evidences of endarteritis and peri-arteritis. The distinctions between gumma and tubercle are the less amount of cheesy degeneration in the centre of the former, its more irregular appearance, the presence of arteritis and vascularization, the absence of giant cells and of tubercle bacilli.

Actinomycosis is a form of infectious tumor which sometimes extends from the face and neck into the brain, leading to inflammatory processes, however, rather than to true tumors. No other neoplasms of infectious origin attack the brain unless glioma be found to be of that nature.

Glioma may occur in any part of the brain, but is most frequently found in the cerebrum. It is the only tumor which is peculiar to the nervous centres, being developed from the neuroglia tissue which forms the supporting structure of these centres. Glioma originates in the white matter of the nerve centre and not from the membranes or fibrous structures. It may grow to a very large size and is the form of brain tumor which becomes the largest. Gliomatous tumors measure from three to eight or more centimetres in diameter. In appearance the glioma can be scarcely distinguished from the brain substance itself, but usually looks like either pale or congested gray matter, or it may have a yellowish or gelatinous appearance. The tumor is very vascular and it may show the results of hemorrhages. The central part sometimes breaks down, forming cavities or cysts. The tumor may grow very rapidly, infiltrating the normal tissue. In these cases there is hardly any definite boundary between the tumor and the normal tissue. In other cases the tumor grows slowly, but rarely if ever becomes encapsuled. Microscopically it is found to consist of small cells with delicate fibrous prolongations, these being the glia cells. The tumor is very vascular and its whole appearance is suggestive of an inflammatory process rather than a new growth; the inflammatory process being one in which the neuroglia tissue reacts to the inflammatory irritant. Gliomata may undergo certain changes, *e.g.*, a mucous degeneration of the cells takes place, forming a myxo-glioma. When there is with the neuroglia-cell proliferation a rich proliferation of round cells from the connective tissue it is called a glio-sarcoma. When the tumor is situated near the surface, involves the membranes, and

grows slowly, with an increase in fibrous tissue, it is called a fibro-glioma. When the gliomatous growth is very firm and hard, the fibrous portion of the glia tissue predominates; it constitutes a nodule such as is found in multiple sclerosis, and these hard gliomata are sometimes called neuro-gliomata.

Sarcoma. The sarcoma and its various modifications form perhaps the most important and almost the most frequent of the brain tumors. The sarcoma is a tumor of connective-tissue origin; it develops, therefore, from the brain membranes or from the sheaths of the blood-vessels. Sarcomas may be single or multiple. They may be of all shapes and they grow to very varying sizes. They often develop a capsule. They may be either primary or secondary. Their growth is often rapid. They are white or grayish in appearance or may be somewhat yellowish, dependent on the predominance of the different kinds of cells and blood-vessels. Microscopically they are made up of small round cells, spindle cells, and other cells of various sizes and forms. They contain often considerable fibrous tissue. They contain blood-vessels, but are not richly vascular. The essential characteristic of the sarcoma is the rich development of round cells and spindle cells; in other words, its rich cellular contents. Sarcomata are peculiar in undergoing many modifications. Thus sometimes fibrous tissue develops largely and the tumor is called a fibro-sarcoma; sometimes the tumor undergoes mucous degeneration and is called a myxo-sarcoma. There may be a breaking down of the centre with the formation of cysts. There may be a development of pigment. Not infrequently a sarcomatous process invades a glioma and we have a mixture of a sarcoma and glioma. Sarcomatous tumors sometimes have an alveolar structure. These tumors contain endothelial cells derived from the lymphatics and are called endothelioma. When sarcomata develop from the dura mater and are slow in growth there may be calcareous deposits in them and they are called psammomata.

The fibroma is a very rare brain tumor, unless the pacchionian bodies, when enlarged and hardened, may be so considered.

Osteoma is not particularly rare, developing in the form of bony plates in the dura, falx, or tentorium. Osteomata in the brain substance are mere pathological curiosities.

Enchondromata, lipomata, and angiomata are rare and have no practical importance.

Occasionally neuromata or false neuromata are found developing on the roots of the cranial nerves.

Cancer is relatively a very rare affection of the brain, especially as a primary development. It usually arises from the membranes

of the brain. Cancer is not infrequently multiple and is usually of the soft or colloid character.

Parasitic growths. Parasitic tumors are extremely rare in this country. The only forms which are found are the echinococcus and the cysticercus cellulosæ. The echinococcus produces hydatid cysts, which may be large or small, few or many, and are usually all upon the surface of the brain. They are much rarer than the cysticerci. These form cysts which are usually multiple, slow in growth,

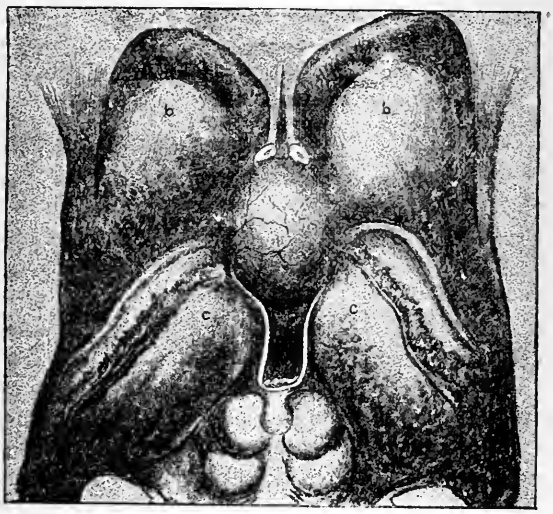


Fig. 216.—Echinococcus Cyst of the Third Ventricle.

lie upon the surface of the brain or in the ventricles, are encapsuled, and show no symptoms (Fig. 216).

Aneurisms are anatomically tumors, but clinically they present some special symptoms and hence are described separately.

Diagnosis.—It is necessary first to make the diagnosis of the presence of the tumor, next of its location, and finally of its nature. The existence of a brain tumor is determined by the presence of the characteristic general symptoms—headache, vomiting, vertigo, optic neuritis, mental disturbances, and progressive course. The physician must bear in mind the possibilities of meningitis, abscess, lead poisoning, hysteria, and paretic dementia. Very often a localized basilar meningitis of syphilitic or tuberculous origin simulates closely the presence of a tumor. Besides the general points referred to, in estimating the probabilities of the existence of a tumor we must bear in mind the age of the patient and the existence of a tuberculous or syphilitic history, the history of an injury, of local tender-

ness, and of rise of cranial-surface temperature, and the presence of some new growth in other parts of the body, particularly about the neck or thorax or in the lungs.

The diagnosis of the location of the tumor is based upon the rules already given in regard to local diagnosis. The diagnosis of the nature of the tumor can often be made and should be attempted. In children, for example, the chances of the tumor being tuberculous are very great, particularly if there is a scrofulous diathesis or tuberculous disease elsewhere. Syphilitic tumors of the brain are almost always accompanied by or preceded by manifestations of external syphilis. Gliomatous tumors occur in childhood and early life. They produce, as a rule, fewer irritative phenomena and are accompanied by remissions and by apoplectic or pseudo-apoplectic attacks due to the vascular nature of the tumor. Carcinomata occur late in life and are usually secondary.

Prognosis.—In extremely rare cases tumor of the brain appears to stop growing and become encapsulated and atrophied. Such tumors are of a tuberculous or syphilitic, perhaps sometimes of a sarcomatous character. As a rule, the brain tumor grows steadily and the symptoms of the disease become more pronounced until death occurs. The prognosis is best for tubercle in children and gumma in adults. It is worse in those cases of glioma and sarcoma which have a rapid course. In fact, the sooner serious symptoms develop, the more rapid the general development of the disorder, the sooner does a fatal termination come. The disease lasts on an average two or three years, ranging from a month to eighteen years.

Treatment.—Something can be done in cases of tuberculous tumors, syphilitic tumors, and possibly in the sarcomatous variety. In tuberculous tumors a general constitutional and strengthening treatment must be resorted to; fresh air, tonics, and a large amount of food being the main reliance. The utility of any form of tuberculin is as yet doubtful. In syphilitic tumors much can be done by the usual vigorous antisyphilitic treatment. In sarcomatous tumors, if they are suspected, some help may be obtained from the internal use of arsenic. Symptomatically we must give such drugs as antipyrin, phenacetin, antifebrin, codeine, and perhaps morphine for the relief of pain. The ice cap and leeching often help the headache also. Should convulsions develop the bromides should be used, just as in idiopathic epilepsy.

In cases in which the location of the tumor can be made out, the question of surgical interference should be considered. The percentage of cases in which surgery can help is extremely small. It will include only those cases in which the tumor can be located; of those

which can be located, only those which are in an accessible region, and finally, of those which are in an accessible region, it includes those which are either superficial, or, if lying in the brain substance, are more or less encapsulated. The removable tumors of the brain amount to less than five per cent. They are, in particular, the sarcomatous, syphilitic, and tuberculous tumors lying in the central or occipital areas. In many cases in which there is some doubt as to the localization an exploratory trephining is justifiable, and in a few cases in which it is known that the tumor cannot be removed the trephining for the simple purpose of relieving pressure is justifiable. It is probable that in adults some tumors from the anterior and middle fossa of the brain can be removed. In children tumors cannot be removed successfully from the cerebellum; in adults it is possible. Operations for tumors should be undertaken as early as possible; this is a fact on which too much stress cannot be laid.

Intracranial Aneurisms.

Intracranial aneurisms are of two kinds—"miliary" and those of large size. The miliary aneurisms are minute dilatations of the vessels and are always multiple; they have been described under the head of cerebral hemorrhage. Large aneurisms affect only the large cerebral arteries at the base of the brain. The arteries are affected in the following order: middle cerebral, basilar, internal carotid, and anterior cerebral. The anterior and posterior communicating and vertebral arteries are occasionally involved, the posterior cerebral and inferior cerebellar very rarely (Gowers).

Etiology.—Males are affected slightly oftener than females. Aneurisms occur at all ages from ten to sixty; before ten and after sixty they are extremely rare. Heredity occasionally plays a part in predisposing to cerebral aneurisms. The exciting causes are embolism, especially when the emboli contain microbes; syphilitic disease, injuries, and in rare cases senile degeneration.

The *symptoms* are very indefinite; they resemble to a considerable extent those of tumor at the base of the brain; headache and vertigo, mental dulness and irritation, cranial-nerve palsies, and occasionally hemiplegia and convulsions are noted. Optic neuritis is rather rare. In a few cases the patient is conscious of a murmur or recognizes the pulsating sensation in the head. Sometimes when the aneurism is in the vertebral artery a murmur can be heard between the mastoid process and the spinal column (Moser).

The *diagnosis* is often difficult; it is based on symptoms of tumor at the base of the brain pressing on cranial nerves and on motor or sensory tracts. The effect of carotid compression should be tried.

The *prognosis* is not good. In perhaps the majority of cases a rupture of the vessel occurs in a few years; however, rupture is not the inevitable event, and sometimes the disease becomes stationary or undergoes spontaneous cure.

The *treatment* of the disease, if it can be recognized, is the same as that for aneurism elsewhere so far as drugs are concerned; surgically the common carotid may be tied and perhaps the vertebral if the aneurism is believed to be connected with that artery or with the basilar.

Syphilis of the Nervous System.

Syphilis is an extremely important factor in the causation of the organic diseases of the nervous system. Nervous syphilis makes up over ten per cent of all the hereditary forms, and while in adult life the specific virus attacks nerve centres relatively less often, yet it is a factor whose importance is very great. In the previous descriptions of nervous diseases we have referred to the syphilitic element in connection with etiology and pathology; but syphilis produces upon the nervous system certain forms of disease which are characteristic, hence it is best to take a brief survey of the effects of this infection independently. Syphilis is beyond much question the result of an infection by a microbe, and the result of the activity of this microbe upon the nervous centres is to produce a condition which is really a form of inflammation. The reader can best understand syphilitic disorders, therefore, by remembering that they are all forms of an inflammation. The syphilitic infiltration is simply an exudative inflammation with a specific exudate; the syphilitic gumma is a deposit of this exudation analogous somewhat to an abscess. Syphilis attacks chiefly the membranes of the brain and spinal cord and the blood-vessels of these organs; in particular the base of the brain and the blood-vessels that supply this region are affected. Syphilis acts on the nervous centres in four ways: it produces meningeal exudation and inflammation, it forms gummatous tumors, it causes arteritis, and it leads to degenerations.

The first three processes are, however, only different types of exudative inflammation, so that we have in reality:

1. Exudative syphilis { meningitis. gumma. arteritis.
2. Degenerative syphilis { brain and spinal degeneration.

Etiology.—A neuropathic constitution probably predisposes to the development of nervous syphilis. The age at which it occurs most frequently is between twenty and forty, but it may occur at all periods of life from infancy up. It attacks men oftener than women in the proportion of about seven to one. Hereditary syphilis makes up about three per cent of the cases. Inadequate and improper treatment of the disease at first probably favors the development of nervous syphilis later. The use of alcohol, excesses in the way of severe bodily exercise, severe mental strain, and overwork predispose to the development of the disease; injuries undoubtedly have a similar effect. The time after the infection when the disease is most apt to occur is the third year, but it is not infrequent between the second and the tenth years, and it is possible for nerve syphilis to develop from within a few months up to thirty years after the infection.

Symptoms.—Since syphilitic changes may attack any part of the nervous centres, the symptomatology of nerve syphilis is necessarily a varied one. The reader can perhaps best understand the way in which the disease acts by having presented first a series of tables showing on the one side the clinical symptoms, on the other the chief anatomical changes that underlie them. The first table presents the symptoms of syphilis of the brain, which is unquestionably the most common form. The next table shows the symptoms of syphilis of the cerebro-spinal system, a form which ranks second in frequency. Third we have syphilis of the spinal cord alone, which is somewhat rarer, and last and rarest of all we have syphilis of the nerves. In addition to these four forms of syphilitic manifestation we have two diseases which are acknowledged to be sequelæ of syphilis and which are called post-syphilitic degenerative processes or degenerative syphilis.

I.—Syphilis of the Brain.

Clinical Symptoms.	*Anatomical Change.*
Severe headache, vomiting, vertigo, mental dulness, and irritability, attacks of somnolence or coma, convulsions, cranial-nerve palsies, optic neuritis, hemiplegia, polyuria, and polydipsia.	Gummatous inflammation of the base involving nerve roots, or gummatous inflammation of convexity, arteritis, and phlebitis.

II.—Cerebro-Spinal Syphilis.

Many of the brain symptoms as above, spastic paraplegia with spinal pains and involvement of sphincters.	Gummatous basilar meningitis; diffuse, disseminated, or localized meningo-myelitis.

III.—Spinal Syphilis.

Paraplegia with pains, Brown-Séquard paralysis.	Meningo-myelitis, gumma, localized softenings from obliterative arteritis.
Spastic paraplegia and ataxia.	

IV.—Syphilis of Nerves.

Cranial-nerve palsies, cauda-equina symptoms, local palsies of peripheral nerves.	Root neuritis, gummatous neuritis.

V.—Post-Syphilitic Degenerative Processes.

Locomotor ataxia; general paresis.

Taking up these different forms of nerve syphilis in order, I will give some further details with regard to each of them.

I. Syphilis of the brain in its most common form shows itself by a gradual development of severe and persistent headache. This is usually associated with vertigo, sometimes with nausea and vomiting. After the headache has developed and has lasted for a time, or even without much delay, there comes on sometimes an attack of hemiplegia. Preceding the hemiplegia, or in some cases without the hemiplegia, there are paralyses of the cranial nerves, more especially of the nerves of the eye. Optic neuritis is somewhat frequent. There may be, before any paralyses develop, attacks of epileptic convulsions, either general or partial. Without any paralyses or with simply cranial-nerve paralyses there may develop attacks of somnolence and coma. Even if such attacks do not appear the patient often shows a mental irritability and weakness, a slowness of the reasoning process, and incapacity to fix the attention such as is observed in connection with brain tumors, only with nerve syphilis these symptoms are not usually so marked. Polyuria and polydipsia are symptoms which are occasionally met with. It will be seen that the syphilitic poison produces very various manifestations when it attacks the brain. The characteristic features are this variability in the symptoms and their remittent character. Elaborate systems of clinical classification might be made out of these various groups, but it will be sufficient for the present purpose to call attention to the fact that the intense headaches, optic neuritis, cranial-nerve palsies, attacks of somnolence and coma, and hemiplegia associated with some of the foregoing symptoms are characteristic of most of the forms. The reason for the peculiar symptoms in brain syphilis is manifest when it is known that the lesion most commonly found underlying them is a gummatous meningitis which has a special predilection for the base of the brain. In particular it seems to attack the interpeduncular space and the neighborhood of the optic chiasm and the surface of the pons Varolii.

This gummatous meningitis consists of a syphilitic inflammatory exudate which surrounds, presses upon, and injures cranial nerves, attacks the arteries of the base, producing an obliterating arteritis and consequent softenings, with the hemiplegia which is so often a manifestation of the disease. Much less frequently the inflammatory process attacks the convexity, and then it assumes the form of a gummatous patch which produces cortical irritation with headaches, mental disturbances, and convulsions.

II. The next form of nervous syphilis is the cerebro-spinal. In this we have almost exactly the same conditions and symptoms so far as the brain is concerned; but in addition there are symptoms due to more or less diffuse syphilitic inflammation of the pia mater of the spinal cord. The syphilitic process often extends into the spinal cord, producing an obliteration of the arteries and softening with the symptoms of a transverse or a central myelitis. Thus we have combined the symptoms of cerebral syphilis and paraplegia with, as a rule, considerable pain in the back, produced by the involvement of the meninges.

III. The third type of syphilis is the spinal form. The symptoms in spinal syphilis are usually those of a transverse myelitis, involving, in the writer's experience, most often the lower part of the dorsal and upper part of the lumbar cord. This myelitis usually comes on rather slowly with the ordinary symptoms of a chronic or subacute transverse myelitis, there being a progressive paraplegia with spasticity of the legs and a good deal of pain. The condition is known as syphilitic spinal paralysis. It is probable that syphilis is a much more frequent factor in the production of so-called transverse myelitis than is usually supposed. The anatomical process underlying it is that of a meningitis which passes along the septa into the substance of the cord, involves the arteries of the cord, and produces a more or less complete softening of the part. The only truly inflammatory process, therefore, is that which is produced in the meninges, connective tissues, and arteries. The anatomical changes in the cord substance are mainly those of softening with reactive inflammation. Spinal syphilis may show itself also by the development of gummatous nodules which grow from the meninges, press upon the cord, and produce the symptoms of a spinal tumor. Spinal syphilis may also develop itself in three or four different foci, producing the symptomatology of disseminated myelitis.

IV. Syphilis of the nerves. Syphilis rarely affects the peripheral nerves; there are, however, occasional deposits of syphilitic exudate producing the ordinary symptoms of irritation and compression of nerves. There is said to be a form of multiple neuritis

produced by syphilis, but its actual existence has not yet been absolutely demonstrated. Syphilis has been known to attack the roots of the cranial nerves, producing a root neuritis; and it is very apt to attack the roots of the spinal nerves when the spinal membranes are involved.

V. The post-syphilitic degenerative processes are locomotor ataxia and general paresis. Occasionally it happens that the syphilitic deposits in the spinal cord may produce lesions somewhat like those of locomotor ataxia, and in this case there will be a train of symptoms which also resemble this disease. In true locomotor ataxia, however, the process is a degenerative not an exudative one. The syphilitic poison seems so to affect the nervous centres as to predispose them to the peculiar degeneration characteristic of tabes. Syphilis may also produce a chronic meningo-encephalitis which will manifest itself by symptoms resembling to a considerable extent general paralysis; but it is very generally conceded that true general paresis is not a syphilitic disorder. Syphilis, however, seems to predispose to it, just as it does to locomotor ataxia.

Hereditary Syphilis.—Inherited syphilis will lead to anatomical changes and clinical manifestations resembling in all respects those of acquired syphilis. Inherited syphilis, in other words, may produce headaches, cranial-nerve palsies, hemiplegia, epilepsy, mental disorders, and paraplegia. The disease probably is the cause of a considerable proportion of the cases of chronic hydrocephalus and of many of the cases of so-called tuberculous meningitis. The peculiarities of hereditary syphilis show themselves rather more in diffuse symptoms such as would be attributed to a meningitis of the convexity; in other words, convulsions and mental weakness are rather more frequent, while hemiplegia and cranial-nerve palsies are comparatively rare. Hereditary syphilis also very rarely indeed attacks the spinal cord, although it is not unlikely that it is a factor in the production of some of the hereditary diseases of that organ. Hereditary syphilis develops at any time from birth to the eighteenth year, but most commonly under the age of five years.

Pathology.—I have already given some indications of the pathological changes produced by syphilis. The disease affects the nervous system (1) by producing a meningitis with infiltration, (2) by producing gummatous masses, (3) by producing an inflammation of the arteries, and (4) by so influencing the nervous system as to lead to the development of degenerative diseases. Of all these forms of anatomical change it is the arteries that are most often affected, and particularly the arteries at the base of the brain.

Syphilitic meningitis is characterized by the proliferation of round cells and the preponderance of an exudate which has a ten-

dency to infiltrate into the nervous tissues. The anatomical characteristics of the syphilitic gumma must be studied in special textbooks. The inflammation of the arteries attacks first the external coat and adventitia, producing there an enormous multiplication of round cells. The external coat becomes weakened, and as a result there develops beneath it, between the intima and the elastic layer, another exudate which constitutes what is known as endarteritis. In syphilitic arteritis, therefore, there is both a peri-arteritis and an endarteritis; the former being usually the primary and most essential process. The endarteritis, however, as it develops gradually produces an occlusion of the arteries. This cuts off the circulation of the blood and leads to softening of the part. There is also a development of a hyaline degeneration in the arteries, which some regard as a very essential part of the anatomical change.

Diagnosis.—The diagnosis of nervous syphilis is based upon the history of an infection, the irregularity and fugacity of the symptoms, the intense headaches, the presence of an optic neuritis, the age of the patient, and the results of treatment. In estimating the importance of the history of infection, it should be remembered that the third year after infection is the serious one for the development in particular of those symptoms produced by obliterating arteritis. In hereditary syphilis the presence of the Hutchinson teeth, the hazy cornea, and deafness or other ear trouble help us in diagnosis. The headache of syphilis is rather characteristic. It may attack any part of the head, but is usually unilateral or irregular, or again it may be bilateral in its distribution. The pain is very intense and sometimes exhibits a certain periodicity. It is usually worse at night. It is apt to last continuously for from five days to three or four weeks. Headache of this character, followed by the paralysis of one or more cranial nerves or by an attack of hemiplegia, is extremely suggestive of syphilis. Optic neuritis is very liable to occur when the disease shows other evidences of being situated at the base of the brain. This optic neuritis is associated with contraction of the visual field, and a characteristic feature of this contraction is that it varies a great deal from week to week. The sex and age of the patient may be taken into consideration in weighing the evidence, and finally the prompt effects of the use of iodide of potassium should have very decided weight.

Prognosis.—It is very difficult to give definite facts regarding the prognosis of syphilis. Unquestionably the outlook is much more favorable than it is for any other organic disease of the nervous system. When the syphilitic process has not produced so much arterial disease as to lead to obliteration of vessels and softening, a

very great degree of improvement and even a recovery may be expected. So far as injuries to the nerves or nerve roots go, we can generally expect a great improvement or cure. Lesions of the convexity are usually amenable to treatment. Syphilitic hemiplegia has a not much better prognosis than hemiplegia from other causes. Syphilitic myelitis has a not very good prognosis, but it is better than that of myelitis due to trauma. Nervous syphilis may last from one to three or four years. The effects of the disease may, if nerve tissue is destroyed, last a lifetime.

Treatment.—As regards the prophylaxis, it is important that persons who have become infected by syphilis should be treated with iodide of potassium in the second as well as the third stage of the disease. After the first year at least, the patient should not neglect to take a certain amount of iodide of potassium four times a year, each course of treatment lasting six weeks. The patient should be warned against indulging in alcohol, against all excesses, mental as well as physical. A laborious life full of worry and anxiety, in which the patient attempts to help himself along with stimulants, is surely provocative of nervous syphilis.

The treatment of the disease when it has appeared consists mainly in the administration of iodide of potasium or sodium. This should be given in beginning doses of ten grains three times a day and increased gradually until the maximum amount which the patient can bear is taken. This maximum is usually between three and four hundred grains a day. In some cases it is important to give more than this—as much, that is to say, as two hundred grains three times a day, and it is the general experience of American neurologists that results can be obtained by these large doses which cannot be obtained by smaller ones. In my own experience I have known a patient to take five hundred grains three times a day for a considerable time without harm, and indeed with benefit. Usually, however, such extraordinary doses are rarely needed. It is found that, as a rule, patients tolerate large doses of iodide quite as well as smaller ones, and sometimes the iodism produced by small doses disappears when large doses are given. The drug is best administered largely diluted with water or with Vichy or in milk, and taken after meals. Some persons bear it better before meals. It is occasionally advisable to combine mercury with the iodide. This may be given in the form of the bichloride or by an inunction. Other drugs which are of value are the ordinary tonics, such as iron, quinine, and the bitters and mineral acids. Plenty of good food, out-door air, and all those things which will improve the general health of the patient are indicated.

CHAPTER XXI.

GENERAL PARESIS: GENERAL PARALYSIS OF THE INSANE—DEMENTIA PARALYTICA.

General paresis is a progressive disease of the brain running a course of about three years, characterized by abnormal mental symptoms, ending in dementia, associated with physical weakness and certain characteristic physical symptoms.

Etiology.—Paresis, as it is usually termed, is a disease of modern civilization, and, as Krafft-Ebing states it, of syphilization. It was a medical curiosity a hundred years ago; now, it is extremely frequent in our asylums, in neurological clinics, and in private practice. It has become much more common of late years in this country. It is found in nearly all the civilized races of Europe and America, but is rare in Africa and Asia. It affects even the inferior races living among civilized people, and is found, for example, among the negroes of the United States.

It is not a disease which is directly inherited, but the neuropathic constitution predisposes to it, and occasional cases are seen in early life which may be said to be of congenital origin and are due to syphilis or degeneration in the parent. It occurs much more often in men than in women, the proportion being about 5 to 1. The proportion of women is slowly becoming greater. In private practice among the better class the number of women who have the disease is very small. In my own case the number of paretic women is about six per cent in one hundred cases.*

The excessive use of alcohol is a predisposing cause, as is also excessive mental exertion, particularly if combined with emotional strain and excitement. Sexual excesses and abuse are also predisposing causes, but the common view, that the disease is the result of perverted sexual indulgence, is not correct. Syphilis is, no doubt, the most essential of all the predisposing causes, and paresis must be put down, with tabes dorsalis, as one of the parasyphilitic

* Among 50 private cases there were 47 men and 3 women (1 to 16). There was a distinct history of syphilis in 23 (nearly fifty per cent). The nationalities were: United States, 27; Hebrews, 10; Irish, 9; Germans, 3; Italians, 1. Total, 50.

diseases, or, as I prefer to call it, one of the degenerative forms of syphilis.

Statistics do not yet give more than twenty to fifty per cent of cases with undoubted histories of syphilis;* in my own experience this percentage is fifty, and the disease is so often and so distinctly traced to syphilis in a large number of cases that we must infer its relationship in the rest. Injuries, sunstroke, exposure, acute diseases may be put down as exciting causes, but their importance is not very great. An almost sure recipe for producing a case of paresis is this: Let a man of nervous constitution acquire syphilis between the ages of twenty and thirty, then let him work as hard as possible without vacation under great mental strain, drink a great deal of alcohol, and indulge excessively sexually. This will be pretty sure to bring on paresis in ten or fifteen years.

The disease occurs most often between the ages of twenty and forty, but it is seen both earlier and later than these ages. It occurs oftener in married men and women. It is seen oftener in the city than in the country.

Symptoms.—In its typical manifestation the disease shows two stages: one of excitement or irritation; the second of dementia and paralysis. In place of the excitement of the first stage the patient may have a prolonged period of hypochondriasis or melancholia, or, with no preliminary stage of excitement or depression, may pass gradually into dementia, the mental and bodily feebleness going on together.

The first type is the most common of all, although the opinion is growing that the types characterized by little active mental disturbance and by more marked physical symptoms are becoming more frequent; in other words, that paresis is becoming more a disease of the brain and spinal cord proper and less a disease of the mind, the mental symptoms† being almost from the first more of a progressive dementia.

Excited Type.—In the first type of cases the patient begins by showing unusual irritability of temper; trivial things annoy him, and his bad humor and change of disposition become noticeable in his family relations and in his business. He is fretful; complains of

*Among personal cases Regis found eighty per cent syphilitic. Krafft-Ebing found, among 175, fifty-six per cent gave a history of syphilis. In 41 cases of paresis in children syphilis could be traced in 87.8 per cent (Zappert). Among 24 cases in adults, 16 gave a history of syphilis (F. Mott).

†Among 40 private cases the types were: excited, 15; hypochondriacal, 10; dementing, 15. Mendel, among 194 cases of all kinds observed since 1880, found 37 of the typical form and 70 of the dementing type.

being easily fatigued; loses interest in his affairs, and is unable to fix his attention for any length of time upon them. He makes occasional mistakes of judgment, and does some extravagant or foolish thing in the way of purchasing or selling. This condition of irritability is followed by one of great mental exaltation. The patient becomes very happy and cheerful and confident; he feels better than he ever did before in his life. He talks excessively, and is effusive and jocose when he used to be sober and reserved. He develops great schemes for the future, he lavishes money uselessly in making presents to his family and friends, or in some extraordinary business venture, and imagines himself possessed of immense wealth or great power. He has what are called delusions of grandeur, or megalomania. This condition of exaltation is interrupted by outbursts of violence, especially if it leads him to indulge in drink, as is often the case. In the course of three or four months the symptoms become so marked that the family recognize the seriousness of his state, and he is confined in some institution where he can do himself and others no harm. Under institutional *régime* he now becomes somewhat more quiet; his exaltation softens down. His symptoms may even remit, and for a time he becomes nearly or quite rational. But after some months he begins to show distinct signs of dementia; the memory becomes weak, he forgets recent events, mislays things, makes mistakes in his accounts, is unable to add correctly; he cannot write a letter coherently, or if he does there are mistakes in spelling and elisions of letters.

During the period of exaltation there gradually appear physical symptoms which are very characteristic. The patient's hands become tremulous, and his handwriting is so affected that his signature often cannot be recognized. There is distinct and decided facial tremor, particularly apparent if the patient is made to close the eyes and stretch the muscles of the lips so as to show the teeth.

There is marked tremor of the tongue, all this tremulousness being much more exaggerated than is seen in other diseases, except occasionally in acute alcoholism. The speech becomes stuttering and thick, and he cannot pronounce long words clearly. On examination of the reflexes it is usually found that the knee-jerks are exaggerated. The pupils are almost always uneven, and, as a rule, react badly to light though fairly well to accommodation, showing, in other words, the Argyll-Robertson pupil; sometimes they do not react either to light or accommodation. The fundus oculi is normal.

There is an early and decided weakness of the sexual function. The bladder may also become weak. The appetite and vegetative organs remain in fairly good condition. The patient often suffers

from persistent insomnia. During this time he also has occasionally vertiginous, syncopal, or apoplectiform attacks. In the latter he falls down and perhaps has hemiplegia lasting for a few days or a few weeks. An epileptic convulsion may occur.

In some cases the knee-jerks are abolished, and there are some ataxia and evidence of a posterior sclerosis. The general muscular power is much diminished, and the patient is unable to take long walks or do any great amount of physical exercise.

In the second stage the most striking feature is the gradual onset of dementia. The patient now becomes more quiet and is inclined to sleep during the daytime. He takes little interest in affairs about him, is extremely forgetful, and is often unable to recognize even his intimate friends. He no longer knows the day of the month nor the year, and cannot tell one anything about current events of the day. He becomes gradually careless about his person, and has to be watched while at his meals lest he spill food on his clothes, and at the toilet lest he soil himself. Finally, he needs to be cared for as if he were a child.

At this late period again, attacks of an apoplectiform character may come on, leaving him temporarily or perhaps permanently hemiplegic. His appetite often continues good, sometimes voracious, and he may gain flesh. He is apt at this time to have periods of excitement at which he has delusions of persecution, or he may have some slight delusions of grandeur. One patient of mine used to weigh himself every day and think he was gaining ten pounds each time. He kept on till he thought he weighed nine hundred pounds.

In the last scene of all he becomes bedridden and helpless, and finally dies of exhaustion. The somatic symptoms during this last period consist of increased tremor, disturbances of speech, and gradual muscular weakness until the patient becomes helpless.

The average duration of the disease is about three years. There are some acute, galloping forms in which the patient dies within a year, and there are some cases in which the patient reaches a stage of partial or complete dementia and remains in this condition for ten or fifteen years.

The Hypochondriacal Type.—In this form the disease begins with symptoms resembling those of neurasthenia and hypochondriasis. The patient complains of disagreeable sensations about his head, hemicrania, pain in his limbs and back, inability to sleep, disorders of the stomach, and vague sensations of discomfort and oppression which he is unable distinctly to describe. These patients are often treated as neurasthenics for a long time, and at first show hardly any physical or mental symptoms suggestive of the real trouble.

Careful examination, however, almost invariably reveals rigid pupils, or a history of syphilis which should always put one on guard.

After a period of perhaps a year, evidences of mental disturbances begin to appear, and they are mostly those of dementia with perhaps delusions of persecutions and suspicion. These delusions may be accompanied by occasional outbreaks of excitement and violence, but the paretic is rarely homicidal, and, it may be added, rarely suicidal. After dementia has set in the physical symptoms of tremor, scanning speech, and tremulous handwriting all become noticeable, and the final stage resembles that of the other form.

Dementing Type.—In a third type there is a primary dementia. The disease begins without any excitement or any special depression, with symptoms of forgetfulness, lack of attention to business, and incapacity to do work. The patient makes mistakes in his calculations, mislays and forgets things, and soon is found by his employer to be of no use. He is often good-natured, not unhappy, and without distinct delusions of any kind. The somatic symptoms of tremor, fixed pupils, and exaggerated reflexes appear and become finally characteristic. Scanning speech is not always present, or comes on later.*

Syphilitic Pseudo-Paresis.—There are some cases of paresis in which symptoms of exudative syphilis introduce the disease. The patient has at first eye palsies or attacks of hemiplegia with intense headache followed by convulsions. It is recognized that he has a syphilitic exudate pressing upon some part of the brain, either the base or the convexity, usually the former. Under proper treatment this resolves and he gets over the paralysis and the seizures, but it is now found that his mind is slightly affected. He has no delusions perhaps, and no immediate exaltation, but his memory is impaired, judgment weakened, his emotional condition is one of excitability, and he has to give up business and live a quiet, inactive life. If he does this, in some cases the disease becomes arrested, and he remains fairly well for a number of years. Cases of apparent recovery even have been reported, but in my experience dementia finally sets in, though it may not be till six or seven years have passed.

Alcoholic Pseudo-Paresis.—Persons who have for long periods of time continuously and excessively indulged in alcohol may develop,

* The urine in the excitable stage shows abnormal increase of solids; in the quiet and dementing stage abnormal diminution (Laillier). The red blood cells and hæmoglobin fall below the normal, the latter more than the former; most cases show a slight leucocytosis. There is a decrease in the lymphocytes and increase of large mononuclear cells (J. A. Capps).

and generally do, a condition of mental weakness which to a certain extent simulates paresis. If these patients have not had syphilis, however, it is not a true paresis. The patients become weak-minded, feeble in judgment, poor in memory, their moral instincts get out of control, and acts of extraordinary selfishness, bestiality, and besottedness are manifest. They gradually become more demented, and finally enter a condition of dementia if they are not carried off by some intercurrent disease, as is often the case. These cases, however, do not present the physical symptoms of paresis. They do not have the speech disturbances, the paralyses, or the apoplectiform seizures that characterize the true disease, and if they can be kept from alcohol they may remain in a state of partial dementia for many years.

Pathology.—The disease, as I have already stated, is essentially a parasyphilitic one, and is due to a degenerative change which sets in in the cells of the brain as the result of poisoning by syphilis. These syphilitic changes produce thickening of the membranes and arteries of the brain, with proliferation of the perivascular tissue. It is believed by some that the vascular changes precede the cell degeneration. It is, however, a more general opinion that the cell degeneration is primary, just as it is in locomotor ataxia.

Examination of the brain of the paretic shows that the dura mater is thickened and vascular, and the membrane is adherent to the cranial bones. The brain itself has undergone atrophy (two to six ounces), and there is increase of the cerebro-spinal fluid. The pia mater, especially in the fore and mid regions of the brain, is congested, and there is thickening of it and of the arachnoid, and the whole membrane is adherent to the cortex beneath it, which is found to be œdematous.

Microscopical examination shows increase of connective tissue around the blood-vessels of the cortex, thickening of the vascular coats, and degeneration of the nerve cells in all grades. The disease involves not only the gray cortex but the cells and nuclei of the medulla. There are often associated with paresis lesions of the posterior and lateral columns of the cord; in fact, it is not very uncommon to have a certain amount of locomotor ataxia associated with the lesions of paresis. A small percentage (three to five) of the cases of locomotor ataxia end in this disease.

Prognosis.—The prognosis is usually said to be invariably bad. If one sees the patient, however, in the very earliest stage and removes him at once from all forms of excitement, and makes him live quietly for a year, using anti-luetic or tonic treatment, one can sometimes check the disease, at least for a time. I have several

patients who seem in this way to have been apparently cured. When the disease has well entered upon its course it is incurable by any measures as yet known. It is probably true that the disease shows remissions oftener than it used to do, and that it is less refractory to treatment than it was formerly.

Treatment.—As some of the early symptoms of the disease are often associated with a history of syphilitic infection, it is wise to put the patient promptly under anti-syphilitic treatment. At the same time he should be sent to some quiet place in the country or to some institution where he can be made to lead an extremely regular and quiet, even life. These two measures, if applied early, have produced remissions which have lasted for from three to six months, even a year. After the anti-syphilitic treatment the patient should be given various tonic measures, such as the glycerophosphate of lime in doses of thirty grains a day, with iron and strychnine.

The use of a cold wet pack and ice cap applied daily for a period of about one to two hours, followed by massage, sometimes produces decided relief, especially in the more excitable cases. The tonic form of hydrotherapy, such as cold baths and douches, is indicated in the depressed types.

CHAPTER XXII.

FUNCTIONAL NERVOUS DISEASES.

FUNCTIONAL nervous diseases are those in which no definite known anatomical change underlies the morbid phenomena. On this account it is customary to classify them on a clinical basis. We can, however, also make etiological and pathogenic subdivisions. Applying such a method now, we have two broadly distinguished classes: the primary, or degenerative, and the secondary, or acquired neuroses. Such a classification is suggestive and helpful, though not perfectly correct, because several factors often enter into the cause of the same neurosis.

- **Primary degenerative neuroses.**
  - Primary neurasthenia, hypochondriasis.
  - Epilepsy.
  - Hysteria major.
  - Hereditary chorea.
  - General spasmodic tics.
  - Myotonia.
- **Acquired neuroses.**
  - From infectious, autochthonous, and mineral poisons.
    - Chorea.
    - Tetanus.
    - Tetany.
    - Rabies.
    - Tremor.
    - Neuralgia.
  - Exhaustion and shock neuroses.
    - Neurasthenia.
    - Hysteria.
    - Exophthalmic goitre.
    - Occupation neuroses.
  - Acquired degenerative neuroses.
    - Tic douloureux.
    - Local spasmodic tics.
    - Paralysis agitans.
  - Miscellaneous. — Vasomotor, trophic, and sleep disorders.

THE DEGENERATIVE NEUROSES.

EPILEPSY.

Idiopathic epilepsy is a chronic functional disorder characterized by periodical seizures attended by loss of consciousness and usually by convulsions. Mental disturbances may accompany or take the place of the convulsions.

Symptomatic epilepsy is a form in which the periodic convulsive attacks are due to gross organic changes in the brain.

Jacksonian or *partial epilepsy* is a form of symptomatic epilepsy usually, and is characterized by periodic convulsions affecting only certain groups of muscles, and often unattended by loss of consciousness.

Hystero-epilepsy is not epilepsy, but a form of hysteria.

Eclampsia or acute epilepsy is the name given to a single isolated attack of convulsions. It is generally of the symptomatic type.

Idiopathic epilepsy shows itself in three rather distinct types of attacks, viz.: that of severe attacks, called the *grand mal;* that of minor attacks, the *petit mal;* and the rarer larvated forms characterized by acute mental disorder and called *psychical epilepsy* or the psychical epileptic equivalent.

Etiology.—Predisposing causes: Heredity is the most potent of any single influence. A history of epilepsy or insanity is found in the family in about one-third of the cases and rather more on the paternal side. Alcoholism and the intermarriage of neurotic persons contribute powerfully to produce the convulsive tendency in children. Powerful emotions during pregnancy, accouchement injuries, and syphilis have some influence. More cases occur in the country than the city, more in temperate climates, and more among in-bred races. All American statistics (Putzel, Hamilton, Hammond, Starr, and myself) show a slight preponderance among males. European observers find it the other way.

Age.—The epileptic age is between ten and twenty, and still more definitely between ten and fifteen. In three-fourths of the cases the disease begins before the age of twenty; in one-sixth of my cases before the age of five. After twenty the danger of epilepsy is slight, and when it occurs it is usually due to accidental causes, like syphilis, alcoholism, or plumbism. Idiopathic epilepsy, however, may develop even after sixty. The accompanying table shows graphically the relation of age to the development of epilepsy, chorea, and neuralgias.

Exciting Causes.—Exciting causes are not present in the majority of cases. The most important are the occurrence of rickets at the time of dentition, fright, injury to the head, sunstroke, infectious diseases, especially scarlatina, masturbation, alcoholism, and syphilis. Masturbation is a real but rare cause, so also is syphilis. The so-called reflex causes are ocular and auditory irritations, worms, dyspeptic states, dental irritations, lesions involving peripheral nerves. Some American observers put much stress on the importance of ocular ir-

ritations. European writers have laid more emphasis on disease of the ear. Probably the gastro-intestinal tract and genital organs furnish the most important exciting irritations. True idiopathic epilepsy may be brought out by peripheral irritations; more rarely there occurs only a reflex epileptiform neurosis.

Symptoms of the Convulsion.—The patient often feels some premonitory symptoms for a few hours or a day, consisting of general malaise, irritability, or giddiness. The attack begins in about half

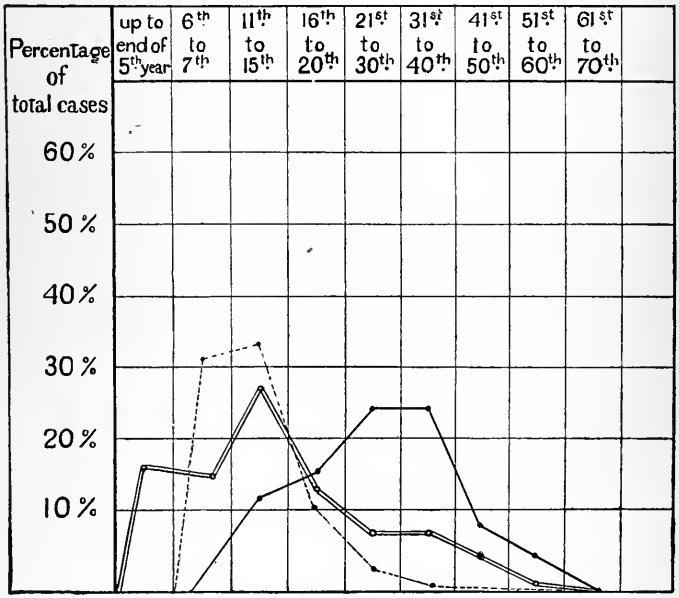


FIG. 217.—TABLE SHOWING PERCENTAGE OF CASES OF EPILEPSY, CHOREA, AND NEURALGIA OCCURRING AT EACH HALF-DECADE AND (AFTER TWENTY) EACH DECADE. Double line, epilepsy; dotted line, chorea; single line, neuralgia.

the cases with a peculiar sensation called the aura. Often also a loud cry is uttered and the patient falls unconscious to the ground. The face is pale, the eyes are open and turned up and to one side, and the pupils dilated. The head is drawn back or to one side, and the whole body is in a state of rigidity or tonic spasm. The arms are slightly drawn out from the trunk, the forearms and wrists flexed, the fingers clinched or flexed in other ways, the legs and feet extended. This tonic stage lasts for fifteen or twenty seconds; the face becomes congested and then livid from compression of the veins of the neck and stoppage of respiration. Gradually jerky movements of the face and limbs begin and the stage of clonic spasm sets in. The trunk and limbs are now alternately flexed and

extended with violent shock-like contractions, the facial and eye muscles twitch, saliva collects in the mouth, and as the tongue is often bitten it becomes stained with blood. The movements are sometimes so violent that the patient is thrown about the bed or floor, and occasionally a limb is dislocated, usually the shoulder. The urine often, and the fæces occasionally, are passed. The temperature is raised $\frac{1}{2}^\circ$ or 1° F., rarely more. The pulse, feeble at first, becomes frequent and tense, and then, as the attack subsides, becomes feeble again. The clonic spasm lasts from one-half to one or two minutes. It subsides gradually, and the patient sinks into a stupor, from which he can be roused with difficulty. This stupor is succeeded by a heavy sleep of several hours and a feeling of hebetude which lasts all day. Vomiting sometimes occurs as a terminal symptom. Immediately after the attack there is a temporary exhaustive paralysis, with loss of knee jerk. The pupils contract again and often oscillate. There may be a slight amount of transient albuminuria or glycosuria. The earthy phosphates are found increased; urea is not. There is a distinct lessening of hæmoglobin in the blood (Féré) and of hæmatoblasts. Sometimes the attack is followed by others, and for hours the patient passes from one convulsion into another. This condition is called *status epilepticus*. It usually lasts less than twelve hours, but may last for one or more days and until finally death occurs from exhaustion. It develops only in the severer types.

Symptoms of the Minor Attacks.—In the minor attacks (*petit mal*) the patient suddenly stops in anything in which he is engaged, the features become fixed, the eyes open, the face is pale, the pupils are dilated, often slight twitching of the facial muscles or of the limbs occurs, and consciousness is lost. In a few seconds the attack is over, and the patient, who does not fall, resumes his work or conversation, being unconscious of what has occurred, except that he has had a "spell." Often there is a warning sensation or aura. This is felt as giddiness, sense of fear, numb sensations of the extremities, flashes of light or blindness, or choking sensations. There may be a cry uttered. The minor attacks are in rarer cases accompanied by sudden forced movements; the patient runs a few steps, or turns round, or makes some automatic movements. This is called *procursive epilepsy*.

Symptoms of the Psychical Attacks.—Sometimes the minor attacks are followed by outbursts of maniacal excitement or by sudden violent automatic movements, and in these states the patient may commit crimes of violence. In rare cases the patient passes into a somnambulic state, during which he performs accustomed

acts, such as driving and walking, automatically and naturally (somnambulic epilepsy). This form of epilepsy may come on without a preliminary minor attack, and then it is to be considered a "psychical epileptic equivalent."

Minor attacks may end in convulsions of a co-ordinate type in which the patient jumps, kicks, throws the arms about as in hysterical attacks. These are called hysteroid convulsions.

The seizure may consist of only a short tonic stage and a few twitchings of the limbs, the whole lasting but a few seconds. This is called an *abortive attack*. Under the influence of medication, the severe seizures are often reduced to abortive forms.

Jacksonian or *partial epilepsy* is a form of the disease characterized by convulsive attacks affecting only a single group of muscles or a limb, and generally not accompanied by loss of consciousness. Jacksonian epilepsy is always symptomatic of some focal lesion affecting the cortical motor area of the brain. This may be a tumor, inflammation, or injury. This form of seizure is particularly significant of a slowly growing brain tumor or syphilis.

The *aura* usually consists of a sensation of numbness, prickling or of a breeze beginning in the hand or leg and passing up to the head, when consciousness is lost. Still oftener there is a peculiar sensation starting in the epigastrium and passing upward. More rarely there are special-sense auræ, such as flashes of light, noises, or voices and peculiar tastes or smells. Besides these there occur feelings of giddiness, dreamy states, peculiar sensations in the head, and indescribable general sensations.

The auræ may be divided into:

Visceral—epigastric, laryngeal, cardiac.

Cutaneous sensations.

Special senses—flashes of light, etc.

Psychical—emotions, dreamy states, etc.

Cephalic—giddiness, etc.

The aura is thought to indicate the seat of the first discharge of nerve force, and its study is of most importance in connection with symptomatic epilepsies, as will be shown later.

Relative frequency of the different kinds of attacks. The severe attacks are the most frequent, next come combinations of severe and minor attacks, and next minor attacks alone, while the psychical forms are the rarest.

Frequency of the Attacks.—The severe attacks may come on only once or twice a year, and this commonly occurs during the development of the disease. The frequency gradually increases until they occur every month, or two or three times a month. Sometimes the

fits occur in groups of four or five every month or two. In very bad cases convulsions occur every day. The *petit-mal* attacks are more frequent and usually occur daily.

Time of Attacks.—The moon and the seasons have no influence. More attacks occur during waking hours than during sleep; but two-thirds of the attacks occur between 8 A.M. and 8 P.M. Many patients have their attacks early in the morning just after awakening (*matutinal epilepsy*). Many attacks occur between 3 and 5 A.M., when the temperature of the body and the vital powers are at the lowest.

State of Patient between Attacks.—Epileptic patients often feel better for a time after the convulsion is over. They not rarely suffer from severe neuralgic headaches; the appetite is capricious, often in children it is voracious, but in older cases there may be anorexia; the bowels are usually constipated; the pulse is small, soft, and frequent in the young, later it is often slow.

Mental Condition.—A gradual mental deterioration occurs in the great majority of epileptics, but it is slight in some and not very great in others. It shows itself by feebleness of memory, irritability of temper, selfishness, incapacity to concentrate the mind or to carry out a purpose. In children great mischievousness and lack of moral sense, with vicious impulses, may appear. The mental deterioration is dependent on those underlying factors which cause the disease. It is apparently in some cases due to the excessive number of the fits. This is not necessarily the case, nor it is generally true that it occurs more often with *petit mal*. It is more marked in cases beginning very early in life, but this is true only when there are decided marks of physical and mental degeneration present. A certain rather small percentage of epileptics become either demented or insane. True epilepsy is not compatible with extraordinary intellectual endowments. Cæsar, Napoleon, Peter the Great, and other geniuses may have had some symptomatic fits, but not idiopathic epilepsy.

Physical Condition.—Epileptics are rather undersized and of not very robust constitution (Féré). They always present some of the marks of degeneration, physical, physiological, or mental. Such marks or stigmata are about ten times more frequent than in healthy persons. The physical stigmata are (Féré) short stature, cranial asymmetry (in 71 per cent), short parietal or frontal arc, and triangular skull; in women high prominent forehead; bad teeth badly placed, high palatal arch; facial asymmetry; prominence of occiput and lemurian hypophysis; differences in color, size, position, and shape of pupils; astigmatism (in 75 per cent of cases); badly shaped

and placed ears; misplaced crown of scalp; low vital capacity; small genitals, atrophic uterus; greater development of left side; long fingers. Cranial deformities of pronounced type occur in epileptics associated with idiocy, hemiplegia, and brain defects of early origin. Sometimes, apparently from a premature ossification of sutures, there are the peculiar shapes of the skull known as scaphocephaly, or steeple skull, and plagiocephaly, or obliquely deformed skull.

The physiological marks of deterioration are a lessened muscular strength (as 35 to 50), habit choreas, a rather imperfect eye with excessive amount of astigmatism and functional muscular weakness. There is a lessened vital capacity, weak and slow digestion, and

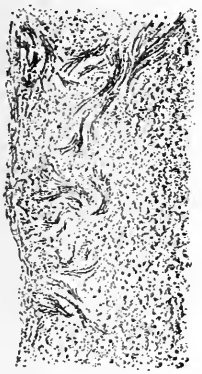


FIG. 218.—DIFFUSE NEUROGLIA SCLEROSIS OF THE CORTEX IN EPILEPSY.

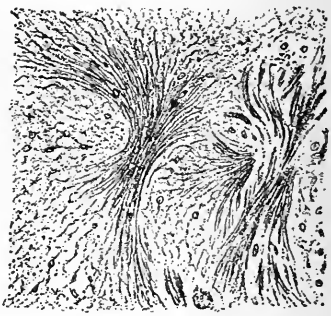


FIG. 219.—SAME, ENLARGED (Chaslin).

sexual atrophy or irritability. The excretion of phosphoric acid is below normal as compared with urea.

The psychical stigmata are mental feebleness, moral insensibility, irritability, wayward and vicious impulses, lack of will power, and sexual aberrations.

Pathology.—The body of the epileptic shows sometimes skin eruptions and ulcers, the result of treatment. There are often evidences of local injuries and fractures due to falls. The organs may show vices of conformation. The uterus is frequently infantile or sharply flexed. Deformation of the occipital bone or the atlas so as to produce narrowing of the upper spinal canal has been noticed. The brain may be unduly large or small, but there is nothing constant in this, nor is there an abnormal difference in the weight of the two hemispheres. The convolutions show many anomalies, but there is in them nothing specific. On the whole the convolutional type is a simple one. The pathological change found most constantly in epilepsy is an induration or sclerosis (gliosis). This

affects the cornu ammonis rather often (4 to 10 per cent), more rarely the olivary bodies or cerebellum. Besides this, small patches of induration occur in the gray matter in various parts of the cortex. Chaslin finds a diffuse increase of neuroglia tissue throughout the brain, more marked when the case is older Fig. 219). Others have found an increase in the neuroglia cells (Kingsbury). In old cases there is often a chronic leptomeningitis, and vascular changes due to the frequent congestions of the brain take place. These consist in varicose and fusiform dilatation of vessels, with evidence of small hemorrhages. Slight degenerative changes in the nerve fibres are also observed.

Bevan Lewis finds in epileptics with insanity a fatty degeneration of the nuclei of the "angular cells" of the second layer of the cortex. In the severer and later stages of the disease this nuclear degeneration is increased so that vacuoles are formed. The cells of the deeper layers are also affected, but to a less extent. The change, though not peculiar to epilepsy, is more extensive and pronounced in this disease.

To sum up: The anatomical basis of idiopathic epilepsy consists in a degeneration of the cortical cells. Also a proliferation and increase in the neuroglia tissue, this occurring most markedly in various islets or special areas of the cortex. The blood-vessels and connective tissue are involved only secondarily and later.

Physiology.—The epileptic fits are due to sudden discharges of nerve force. The seat of the discharge is the cortex of the brain. The discharging cells are, in the severe seizures, the large motor cells, the function of which is to store up and discharge nerve force. They are under control of the sensory cells (angular cells) of the second layer, which have an inhibitory power. These being diseased, their control is weakened and the motor cells "explode" periodically. In sensory and psychical epilepsy the same mechanism exists. The more highly organized cells with large nuclei of the second layer are congenitally or otherwise weak and diseased; the cells below them are not maintained in stable equilibrium and hence periodically break down and "discharge."

The *diagnosis* is based on the character of the attacks and has to be made from hysterical and various toxic and symptomatic convulsions.

The aura, the scream, the quick loss of consciousness, the dilated pupils, the tonic convulsion, the bitten tongue, the emptied bladder, are all characteristic. The hysterical patient sometimes, but rarely, loses consciousness, the epileptic almost always. Hysterical patients do not hurt themselves in falling or bite their

tongue, and their muscular movements, while irregular and violent in character, are yet co-ordinate, *i.e.*, they throw themselves about, kick, strike, etc. Their attacks often are produced by emotion and are ended by some powerful mental or physical impression. The slight rise of temperature in epileptics rarely occurs in hysterics. *Petit mal* and epileptic vertigo are distinguished by the sudden lapse of consciousness and by the sudden pallor and fixation of the eyes, dilatation of the pupils, and slight twitchings of the face. Nocturnal convulsions are usually epileptic.

Eclampsia, or acute symptomatic and reflex convulsions, cannot always be distinguished from epilepsy. The history of the case, the irregular and often prolonged character of the fit, may enable one to make the diagnosis.

Course and Prognosis.—Epilepsy shortens life to some extent; most subjects do not live beyond the age of forty or fifty. About ten per cent become demented or insane. Five or ten per cent get well. The remainder reach a certain stage of severity in their disease and continue in it for years. This severity depends on the treatment, the nature of the attacks, and the extent of degeneration which the organism shows. While unquestionably treatment cures or suppresses the disease in some cases, it disappears spontaneously in others. The prognosis of *petit mal* is worse than that of *grand mal;* that of the two combined is worse still, yet not hopeless. The psychical form of epilepsy is the least amenable to treatment. Epileptic insanity and dementia are incurable. Death occurs rarely in the attacks except in terminal stages. Yet the *status epilepticus* is always a source of danger.

Epileptics are said rather frequently to suffer from phthisis. This is, however, a matter of infection and can be prevented. It should be remembered that epileptics who have only a moderate number of attacks, six to fifteen yearly, can get along comfortably for years, doing their work and enjoying a fair share of the duties and pleasures of life. Finally, the following prognostic rules may be laid down: The prognosis is better in males, better if there is a hereditary history, better if the fits are nocturnal or diurnal alone, better in *grand mal*, better if fits occur infrequently, better if they begin after twenty, and better if due to extrinsic causes. The prognosis is very bad in post-hemiplegic epilepsy and epilepsy due to organic disease.

Treatment.—The first and essential rule of treatment is to take cases early and treat them vigorously from the start. Children who have had a few convulsions during the first three or five years of life should be treated as if they might develop epilepsy between the

ages of ten and fifteen or earlier. The recurrence of a fit between the ages of five and ten should excite apprehension and call for the most diligent treatment. Another rule is that when epilepsy is recognized in children the case should be treated constantly for at least three years after all attacks have ceased.

Constitutional Treatment.—Along with the evolution of epilepsy there is probably a progressive diffuse neuroglia sclerosis of the brain. Whether this is primary or secondary, it is at least proper to use those measures which apparently affect this neuroglia proliferation. Mercury, arsenic, and perhaps iodide of potassium are drugs which we have good reason for believing affect this.

Besides this, we should use measures that increase vasomotor tone and strengthen and steady the circulation. Nothing does this better than water. Epileptics should be given showers, douches, cold sponge baths, or wetpacks according to their needs and opportunities. They should also drink water freely. Again, the nervous system is greatly steadied and quieted by mental occupation that interests one. Nothing is more unfortunate than the idleness often inforced on epileptics. I have seen the disease absolutely checked by having a boy learn a trade that he liked.

The next most important indication is diet, the prevention of intestinal decomposition. In *petit mal* particularly an absolutely non-irritating diet, such as milk, meat, and bread, will quickly lessen or stop the attacks. Meats can be taken in moderation if eaten slowly. As a rule it is a little safer to keep meat out of children's diet for a time; but in adults it is not necessary, though it should be given in moderation.

Removal of irritating causes. Malaria if present promotes the convulsive tendency; so also do lead and acohol; tobacco does not do this, but its use is better stopped, as it is liable to weaken vascular tone and impair digestion. Syphilis causes epilepsy only through producing organic changes.

The rheumatic, gouty, and so-called tuberculous diatheses do not stand in any close relation to epilepsy. The condition known as lithæmia, however, in which there are insufficient oxidation and excretion of products of tissue waste, needs attention. Hence the use of bicarbonate of potassium, salicylate of sodium, the alkaline mineral waters, and a restricted diet are not rarely indicated.

The importance of reflex irritations has been much overestimated. Still they must be considered. The most serious are those arising from the gastro-intestinal tract, the sexual organs, and the eyes. Phimosis if present must be relieved, and masturbation or sexual excesses stopped if possible. It is admitted now that re-

moval of the ovaries, even if diseased, never cures true epilepsy, though it may help hysterical convulsions.

Astigmatism and hypermetropia should be corrected; also ocular insufficiencies if these are pronounced.

Proper attention to the frequent constipation and dyspepsia is of course necessary. The use of hot water is often serviceable, a glass being sipped slowly before the morning and evening meals. This helps also to carry off the bromides and wash out the system generally. Still further to promote this, a purge should be given every fortnight or month. Out-door life and active physical exercise are indorsed by Hippocrates. They do not have any specific influence unless associated with some employment.

Specific treatment. The drugs which have obtained and held a reputation as anti-epileptics are not numerous. They are the bromides, chloral, chloral-amide, belladonna, zinc, nitroglycerin, antifebrin, and antipyrin. Of less value are digitalis, cannabis indica, borax, valerian, and ergot. As adjuvant drugs we have quinine, strychnine, iron, the phosphates, arsenic, silver, the alkalies and iodides. The most valuable of the specific drugs are the bromides.

All bromides act alike in this disease. If one does not cure another will not. Occasionally, changing or mixing reduces the attacks for a time and benefits the stomach. The best bromides are those of potassium, sodium, strontium, ammonium, and hydrogen (hydrobromic acid). Pure bromine may be used.

Bromide of potassium is the most trustworthy. Bromide of sodium is more agreeable to the taste, less irritating to the stomach and milder in its effects, but is eventually just as depressing as other forms. Bromide of ammonium has a brief stimulant effect on the circulation. Bromide of strontium has no advantages that I can discover. Bromide of gold is of no use.

Hydrobromic acid is useful in those cases in which there are indigestion and phosphaturia and an alkali is contraindicated. It produces acne less readily than the alkaline bromides.

Bromides should be given in daily doses of ʒ i., increased gradually until the attacks are suppressed or the dose reaches ʒ iv. to ℥ i. daily. Few patients can tolerate more than this latter dose. Thorough bromidization should be always tried if necessary to stop the fits, and it may be occasionally repeated. But bromidization is sometimes injurious, even making the disease worse, and it must always be employed with caution. When the fits are suppressed the bromides should be reduced, but never entirely stopped for at least two years after the last fit. In most cases, and especially in nocturnal epilepsy, an extra large dose of bromide or bromide and

chloral should be given at night. It is very important that the bromides should be chemically pure (most samples are not), that their use should be continued a very long time, and that their depressing effects should be offset by tonics and all possible roborant measures.

Bromides lessen the fits in from eighty to eighty-five per cent of cases. They do no good or do actual harm, as regards frequency of attacks, in from five to ten per cent of cases. Bromides do no actual good to the patient in a much larger proportion of cases.

To prevent bromide acne, arsenic, calcium sulphide, baths, and diuretics are the best measures.

To prevent bromidization, one should adopt all possible roborant measures; use salt-water baths and regular physical exercise; give black coffee, caffeine, cocaine, mineral acids, strychnine, bitter tonics, cod-liver oil. In all cases the patient should dilute the drug, preferably with carbonic-acid water or Vichy, in the proportion of six ounces of water to a scruple of the drug. A few drops of phosphoric acid may be added to this.

The continuous administration of an alkaline bromide in an alkaline water sometimes affects the bladder, and then the bromide can be given dissolved in hydrobromic acid.

The best substitutes for the bromides, when these do no good or do harm, are belladonna, zinc, strychnine, glonoin, borax, and antipyrin.

The best non-specific adjuvants (drugs) to the bromides are potassium iodide (in syphilitic epilepsy), carbonate and sodium salicylate (in lithæmic and rheumatic states), carbonate of ammonium, the hypophosphites, arsenic, iron, and quinine.

One of the best specific adjuvants to the bromides, as Seguin has shown, is chloral hydrate. By adding five or six grains of this to a mixture the bromide dose can be reduced one-half and the fits still be controlled. Chloral-amide has a similar effect. Both these drugs will sometimes affect the eyes and stomach unfavorably. Other excellent adjuvants are salicylate of sodium and antipyrin. Children bear nearly as large doses of bromide as adults.

The remedies that are especially useful in *petit mal* are, after the bromides, antipyrin, bromide of camphor, belladonna, glonoin, cannabis indica, cod-liver oil, ergot, counter-irritation at the back of the neck, and cold spinal douches.

For epilepsy in children, besides the bromides it is sometimes advisable to employ milk diet, rest, and oxide of zinc.

In hemiplegic and in Jacksonian epilepsy the actual cautery applied over the scalp is beneficial. Urethane occasionally acts well also, but it is of no use in ordinary epilepsy and it may produce

albuminuria if given in large doses. Strychnine is sometimes useful. Raising the head of the bed or making the patient sleep in a chair at night are measures that may be tried.

For hysterical and erethitic cases, with or in place of bromides give a diet of milk and vegetables, and try turpentine, valerian, or zinc. Belladonna is usually contraindicated.

Counter-irritation by means of blisters, issues, and setons at the back of the neck is of doubtful value.

For the status epilepticus give large enemata of chloral and use emetics and purges. Venesection is often efficacious, morphine is dangerous, chloroform is only palliative, and nitrite of amyl is of little value.

To prevent impending attacks the best remedy is nitrite of amyl, which may be carried in a phial filled with cotton. Inhalation of chloroform or ammonia, the internal administration of ammonia, spirits of lavender, or alcohol, a sternutatory, and pressure on the carotids—all are measures which sometimes stop the attack.

Alterative and habit-breaking drugs, such as mercury, iodide of potassium, arsenic, and antimony, are useful in epilepsy, especially in acquired forms due to lead, alcohol, and syphilis. Bromides stop the fits oftener if given early in the disease, if given to young children, and if given in cases that develop after twenty-one.

Injuries to the head which have caused a fracture or a contusion of the brain are the most frequent traumatic irritants. Whenever epilepsy can be distinctly traced to a blow on the head the question of trephining should be brought up. If there is a history of fracture, or present evidence of fracture, or even evidence of severe head injury, trephining is justifiable. The more marked the evidence of a degenerative constitution and the less marked the evidence of real brain injury, the less hopeful the prognosis. On the whole, surgery can do little for acquired and nothing for idiopathic epilepsy.

CHAPTER XXIII.

HYSTERIA.

Hysteria is a chronic functional disorder characterized by nervous crises of an emotional, convulsive, or other nature and by an interparoxysmal state in which certain marks or stigmata are present. Hysteria is essentially a psychosis, and the dominant symptoms are attributable to disorder of the cortical areas of the brain. Its components are the paroxysms, or "crises" as they are called, on the one hand, and the peculiar symptoms of an interparoxysmal state on the other hand. The disease is to be regarded as a definite one, having a certain, as yet unknown, pathological basis underlying it. The use of the word should be much more restricted and definite than has hitherto been the fashion. There are two forms of the disease, hysteria major and hysteria minor.

Etiology.—Of the predisposing causes heredity is the most important. In about seventy-five per cent there is a history of hysteria or some neurosis or psychosis in the parents. The disease is transmitted more often by the mother. Heredity is particularly apt to be important in the hysteria of children; it is a much smaller factor in hysteria of adult males. A hereditary history of rheumatism, gout, and tuberculosis is of very doubtful importance. Hysteria is a disease of early adult life, most cases occurring between the ages of from fifteen to twenty-five in females; it occurs later in males. Hysteria attacks children between the ages of eight and fifteen, chiefly between eleven and fourteen. The disease affects women more than men in the proportion of four to one, varying much with race, climate, and occupation. Hysteria occurs in all classes of life, but rather less frequently in the middle classes than among the poor and the very rich. Male hysteria is more frequent in the poorer classes who are subjected to the exciting influences of alcoholism, poverty, injuries, etc. Hysteria is certainly much less frequent in its severer forms in this country than in some parts of Europe, particularly France. In my experience it is much less frequent than epilepsy in the northern and eastern parts of this country. It occurs, however, quite frequently in the negroes and also in the Latin races of this country. Bad methods of education.

and bad family training undoubtedly tend to promote the development of the disease.

The most important single exciting factor is powerful emotion, particularly fear. Other emotions of an allied character—excitement, sorrow, anxiety—may bring on attacks. The disease can be developed by imitation. Injuries combined usually with mental shock are fruitful causes of producing hysteria. The infectious fevers, syphilis, diffuse hemorrhages, the poisons—lead, alcohol, mercury, and tobacco—the administration of ether, mental and bodily and sexual excesses, are all important agents in developing the disease.

Symptoms.—The symptoms of hysteria are best described under two general heads: first, those of hysteria minor or the hysterical condition, and second, those of hysteria major.

1. Hysteria minor is characterized by the interparoxysmal condition of emotional weakness, nervousness, hyperæsthesia and pains, and by crises of an emotional character. In hysteria minor there are no permanent objective marks like anæsthesia and paralysis, and no decided convulsive seizures. The patient, who is almost always a girl or young woman, gradually develops an undue sensitiveness, the mind is depressed, and she gets easily alarmed. She has feelings of nervousness and lacks control over the emotions, she laughs and cries very easily and yields to every impulse. She suffers from headaches, which are usually vertical and often severe and chronic, and from spinal pains. She sleeps as a rule rather badly and often has disagreeable dreams. She has, under any little excitement, sensations of tickling, fulness or choking in the throat, forming the condition known as globus. Excitement also brings on attacks of trembling or chilly feelings which come and go. There is more rarely a considerable amount of vasomotor instability, as shown by flushings and by coldness of the extremities.

She has with more or less frequency distinct crises of an emotional character, during which she laughs or cries without apparent cause, or at least to an extent beyond her control. She may have attacks of vomiting or headache, or of intense mental excitement amounting almost to delirium. In some cases the patient has somnambulic attacks at night, or she may have under a little excitement attacks of cerebral automatism during which she involuntarily does things that she is entirely unconscious of when she comes out of the attack. The crises are followed by a copious discharge of very light urine. Hysteria minor is closely allied to a condition of neurasthenia or of simple nervousness. It is associated with neurasthenia oftentimes, and is to be distinguished from it chiefly by the peculiar

psychical state, the hyperæsthesia, and the crises which have been described. Even in hysteria minor there is a degree of that peculiar mental condition which will be described later and which is known as suggestibility. Hysteria minor is a disease which belongs especially to childhood and early womanhood. It is very apt to become ameliorated and disappear a little later in life or under the influence of proper treatment, but it may continue or pass into the major form.

2. Hysteria major is characterized by interparoxysmal manifestations of anæsthesia, paralyses, contractures, tremors, peculiar mental conditions, and by paroxysms of an emotional, convulsive, or other serious nature. Hysteria major is what is usually meant when one speaks of hysteria; it includes also hystero-epilepsy. The onset may be gradual, but not infrequently it follows some shock, the first symptom being a convulsion, a paralysis, or some emotional outburst.

The *symptoms of the crises* are the most striking and will be described first. The most common of the paroxysms of hysteria are emotional outbursts of crying or laughing; after this come motor disturbances in the shape of convulsions of various types or of hemiplegia or other type of paralysis. Besides this we have attacks of severe pain, forming neuralgic crises; attacks of nausea, gastralgia, and vomiting, forming gastric crises; much more rarely there are prolonged attacks of hysterical coughing, hiccoughing, sneezing, or rapid breathing. The hysterical seizure may also take the form of attacks of trance and lethargy, catalepsy, amnesia, and cerebral automatism.

The emotional crises are characterized by appearing without any good cause; the patient laughs without reason, and the laughing continues and is quite beyond her power of control. In the same way, and rather more frequently, crying attacks or attacks of furious anger and excitement come on. Associated with these outbursts there is almost always a peculiar sensation of something in the throat. It is described sometimes as being a ball or pressure or a squeezing sensation. It is called hysterical globus, and is due usually to a paræsthesia of the nerves of the throat and larynx, but occasionally there is also a muscular spasm of those parts. Following the crises there is a profuse discharge of pale, limpid urine.

Hysterical convulsions have two rather well-defined types. One of them is that which comes on also in hysteria minor and is the ordinary form of hysterical convulsions; the other is a much more severe disturbance in every way and is known as a hystero-epileptic or hysteroid attack. In the hysterical convulsion the patient, under

the influence of some excitement, injury, or acute gastric disturbance, rather suddenly falls down and begins to go through various irregular movements of the body, such as thrashing with the arms, kicking with the legs, throwing the head from side to side, rolling about on the bed or floor. In the more distinctively convulsive seizure the hands and arms and fingers are flexed, the legs and feet are extended, the eyes are generally closed, the eyeballs often converged or moved about irregularly, the pupils dilated. There is some lessening of sensation over the body and of the conjunctivæ. The patient often utters noises or screams at intervals. She may bite her lips, but does not bite the tongue, nor does she ever hurt herself in her various contortions. The attack may last for half an hour to several hours, unless some measures are taken to break it up. In other forms of hysterical convulsion there is simply a general shaking or trepidation of the body as though the patient had a chill; in other cases again the main type of movement is that of opisthotonos, the patient rising up upon the head and heels and arching the body as in tetanus. Again the attack may consist simply of a little rigidity of the body, or of a series of rhythmical movements of the head or trunk or limbs, the patient sitting up and oscillating the head or swaying the trunk or moving the arms, uttering at the same time incoherent words. In still other cases the patient simply falls down and lies unconscious like a person sleeping for a few minutes or even an hour. In children the attacks may be associated with peculiar noises and movements in imitation of animals, such as the growling of a dog or the mewing of a cat. This condition is called therio-mimicry. In some instances the attack may be accompanied by or may end in a condition of mental excitement approaching delirium. The patients while suffering from these seizures generally appreciate what is going on about them, and will often respond to some stern order for them to cease or will be brought to a state of quietude by pressure upon some part of the body which provokes pain. In women in particular, pressure over the ovaries or epigastrium will abort the attack; the application of cold water or an emetic will do the same.

After a hysterical crisis, or sudden shock, the patient may be found to have a paralysis of arms or legs or one side of the body.

The Symptoms of the Interparoxysmal State.—Between the crises the patient may be in a fair condition of general health, but usually presents certain definite chronic manifestations of the disease. The most characteristic are sensory symptoms, paralyses, and contractures.

Sensory symptoms. These consist of cutaneous and mucous

hyperæsthesia and anæsthesia and anæsthetic disturbances of the special senses. Cutaneous anæsthesia occurs in three forms: the common form is that of hemianæsthesia involving one-half of the body; next in frequency is the segmental anæsthesia involving an arm or a leg or part of the face or head; rarest of all the forms is a disseminated anæsthesia occurring in the form of patches. These various modes of distribution are shown in the accompanying figures. The anæsthesia is a pain anæsthesia chiefly. The tactile and thermic sensations are less markedly affected. The anæsthesia is in some rare cases transferable by means of magnets or electrical irritants or by suggestion. The anæsthesia can also be lessened or removed temporarily by the application of magnets or coins or pieces of metal. For example, if a silver coin is fastened upon the anæsthetic area, in the course of a few minutes or a few hours there will be a zone of normal sensation under and around the coin. Sometimes the temperature of the skin upon the anæsthetic part is lowered 3° or 4° F., and upon pricking the skin blood does not flow. The anæsthesia is oftener upon the left side in the proportion of three to one. Hysterical anæsthesias are not accompanied by subjective sensations as are organic anæsthesias. The skin reflex is usually abolished. Anæsthesia of some kind occurs in a very large proportion of chronic forms of hysteria major. They are rare, however, in children, and are rarer in women than in men, in the author's experience. Anæsthesia of the mucous membranes is present chiefly in hemianæsthesia; it then involves the mucous membrane of the mouth and throat, and to a less extent that of the nose and glottis. Hemianæsthesia is usually accompanied by some hemiplegia and often by some tremor. Segmental anæsthesia is also often accompanied by some degree of paralysis of the part.

Visual anæsthesias. One of the most common of the permanent stigmata of hysteria is an anæsthetic condition of the retina. The result of this is the production of a concentric limitation of the visual field and a disturbance in the color sense. Complete loss of this sense may take place or there may be a variation in the ways in which the colors are perceived. There may be also a distinct diminution in the acuity of vision or even a complete loss of sight of one eye. The visual disturbance is most common with hemianæsthesia. It is more marked on the affected side, but exists to some extent on the healthy side. The limitation of the fields is shown in the accompanying cut (Fig. 221).

Hearing. There is sometimes a diminution in the acuity of hearing of one ear, and this occurs, if present, in connection with hemianæsthesia. There may also be a loss of hearing to high

and low notes, while hearing is apparently fairly good to notes of medium range; and finally there may be a diminution in hearing

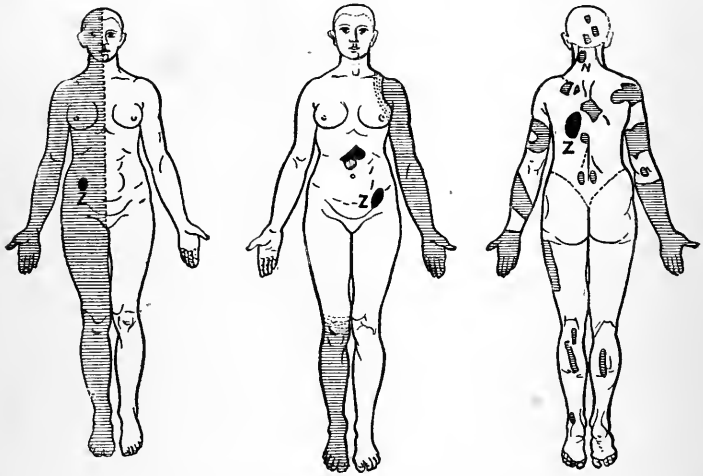
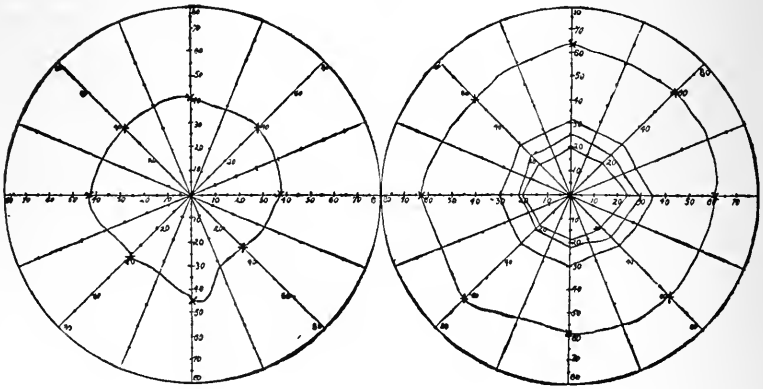


FIG. 220.—THE THREE TYPES OF DISTRIBUTION OF ANÆSTHESIA IN HYSTERIA: HEMIANÆSTHESIA, SEGMENTAL, AND DISSEMINATED. *Z*, hysterogenic zones.

by bone conduction, while hearing by aerial conduction is but little impaired, this being due probably to an anæsthesia of the acoustic nerve.



O. S. O. D.

FIG. 221.—HYSTERICAL LOSS OF COLOR SENSE AND LIMITATION OF VISUAL FIELD. Color sense absent in left eye and field contracted; in right eye field less contracted; order of appreciation of colors from without in was yellow, violet, blue, red, green (observation by Dr. E. S. Peck).

Disturbances of taste in the form of anæsthesia or paræsthesia are quite frequent and are important signs in hysteria. The loss

of the sense of taste may involve only the back portion of the tongue and the palate; in other words, the taste field is here limited as it is with hearing and vision.

The sense of smell may be abolished, but this usually occurs in connection with hemianæsthesia.

Hyperæsthesias and neuralgias. Pure neuralgias are somewhat rare in true hysteria, but hyperæsthesia and pains of various kinds are not at all infrequent. Hyperæsthesia occurs in the form of patches at different parts of the body. These sensitive points may, when pressed upon, bring on paroxysms of various kinds, and they are therefore called the hysterogenic zones (Fig. 220). The most common seat of these zones in women is over the ovaries; in men, in regions corresponding to the ovaries and on the scrotum. Hysterogenic zones, however, may be found just beneath the mammary gland, on the epigastrium, along the spine, and in other places. These zones are sensitive areas; they can be made to disappear by applications of electricity and by refrigeration and counter-irritation. Hysterical patients often suffer from local headaches, which are apt to be confined to the top of the head or to the sides near the temples. The pain is severe, sharp, and boring, and may exacerbate with such intensity as to produce symptoms almost resembling meningitis. The spot-like pains are known as hysterical clavus. Hysterical patients occasionally have migraine, facial neuralgia, and intercostal neuralgia. Much more often they have pains along the spine, producing symptoms of spinal irritation. Hysterical patients also have at times attacks of palpitation and pains over the heart, constituting what is known as pseudo-angina. Such troubles are much more frequent in women.

Motor symptoms. The motor symptoms of hysteria are paralyses, amyosthenia, contractures, tremor, and choreic and ataxic movements. The paralyses of hysteria take the form of hemiplegia, paraplegia, and monoplegias. Hysterical hemiplegia occurs usually rather suddenly, often as the result of some severe shock. The left side is more frequently attacked. The arm is most affected, the leg next, while the face is hardly ever involved. The paralysis is not an absolute one, and the patient is able to drag himself along. The deep reflexes are usually not exaggerated and they may be for a short time absent. The paralysis is thus a flaccid one. The gait of the patient is different from that of hemiplegia due to organic disease; in hysterical hemiplegia the patient drags the paralyzed leg after him, in organic hemiplegia the patient swings the paralyzed leg around in a half circle. This peculiarity of the gait,

the absence of exaggerated reflexes, the absence of paralysis of the face, and the presence very commonly of other hysterical stigmata are sufficient to enable one to make the diagnosis. Sometimes the face on the affected side is slightly drawn by a spasm, so that it appears to be paralyzed when it really is not (Charcot). Monoplegias affect the arm or leg, very rarely indeed the face, occasionally the eye muscles, and most commonly of all the muscles of the larynx. Hysterical monoplegia is usually accompanied by anæsthesia of the affected part and by other symptoms of hysteria. There are no serious atrophic changes or disturbances of the electrical reactions. Hysterical eye palsies show themselves in the form oftenest of an insufficiency of the internal recti, much more rarely by a paralysis of the third nerve or some of its branches. In hysterical palsy of the larynx the adductors are involved so that the patient cannot speak loud, and the condition is called hysterical aphonia. The trouble often comes on suddenly, the patient finding that he cannot speak above a whisper. The paralysis is not so great but that the adductors can be approximated in coughing. The trouble is distinguished from laryngeal inflammation by inspection of the affected part. The abductors of the larynx and the tongue and other muscles of articulation are in very rare cases also involved, and hysteria may produce symptoms resembling a bulbar paralysis. Paraplegia is a rather common form of hysterical palsy; it is usually brought on by emotions of depressing character, often associated with some slight injury. It may be accompanied by a good deal of pain in the back, and the form of disease which is popularly known as "spinal concussion" consists in many cases of hysterical paraplegia combined with hysterical neuralgia of the spine. In hysterical paraplegia there is very little wasting of the limbs and no change in the electrical reactions. The deep reflexes may be somewhat increased or normal; they are never absent. There is never any prolonged or persistent ankle clonus, but there may be a short or spurious clonus due to a general exaggerated irritability of the nervous system. The sphincters are never involved except temporarily or through some complication.

Amyosthenia is a frequent, peculiar, and interesting symptom occurring in the interparoxysmal stage of hysteria. It consists in a more or less temporary feeling of weakness of an arm or of the legs. Thus a person in lifting a dish from the table suddenly feels the arm give out, and if not careful the dish is dropped; or while walking the patients suddenly feel as though they had lost all power in the lower limbs. This amyosthenic condition is generally temporary, but it may be so permanent as to produce a cer-

tain degree of monoplegia or paraplegia. The amyosthenic condition generally precedes a paralysis. It presents no objective signs in the way of electrical reaction; it involves a whole member, not a single group of muscles; it affects more the anæsthetic side, and when it exists the deep reflexes are usually exaggerated (Tourette).

Contractures. In some forms of hysteria there is a tendency for the muscles to undergo contracture under slight mechanical stimulation such as pressure or a blow. This tendency to contracture in hysteria is called the *contractural diathesis*, and it is an important sign. The contractures may be temporary, disap-

Fig. 222.—Hysterical Contracture of the Hand of Six Months' Duration.

pearing soon after the exciting cause ceases, or they may develop independently and last for a long time. They involve the legs, arms, and facial muscles, and may be associated with paralysis and anæsthesia (Figs. 222, 223).

Tremor occurs in hysteria in a considerable proportion of cases, more especially those in which there are hemiplegia and hemianæsthesia. Hysterical tremor simulates all the various types. The common form is one in which the oscillations occur from five and a half to seven and a half times a second, and it is therefore a tremor of slow rhythm. It ceases for a time when the person is quiet or lies in the horizontal position; also during sleep. It affects the head and tongue as well as the extremities, the latter more upon one side than the other. It may be chiefly in the lower limbs. Sometimes it has the type of an intentional tremor, ceas-

ing on rest of the hand and increasing when the hand is moved, as in raising a glass to the lips (Fig. 224). It then resembles exactly the tremor of multiple sclerosis. There may be a slow tremor of four to five and a half oscillations per second; this persists during

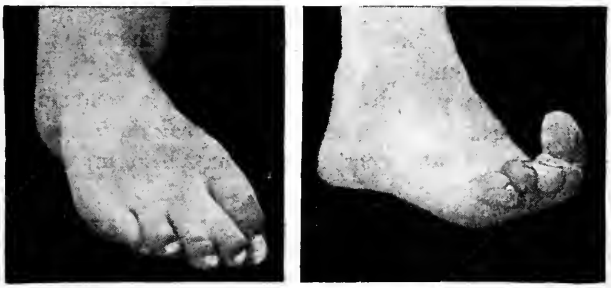
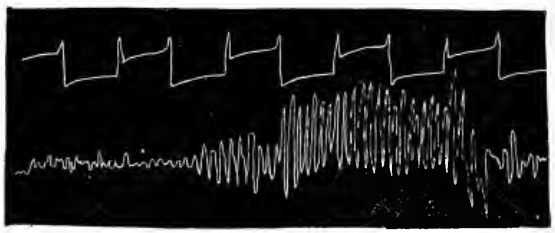


FIG. 223.—HYSTERICAL CONTRACTURES.

rest and is but little modified by voluntary movements. It imitates the tremor of paralysis agitans. Finally there may be a rapid tremor of eight to nine oscillations per second. This also persists during repose and is but little modified by movements. It imitates the tremor of Basedow's disease, alcoholism, and neurasthenia.

The mental state in hysteria is characterized by emotional instability and intense craving for sympathy, weakness of the will, lack of self-control, acuteness of perception, and a constant vari-



REPOSE. MOVEMENT.

FIG. 224.—ILLUSTRATING INTENTIONAL TREMOR IN HYSTERIA (Tourette).

ability of moods. The hysterical mind is not a dull one, it is not a consciously mendacious or evil one. But there are an abnormal increase of sensibility and an exaggeration of the personal feeling or egotism which leads to selfishness and prompts deceit.

The fundamental defect in the hysterical brain is that it is circumscribed in its associative functions; the field of consciousness is

limited just as is the field of vision. The mental activity is confined to personal feelings, which are not regulated by connotation of past experiences; hence they flow over too easily into emotional outbursts or motor paroxysms. The hysterical person cannot think.

Physiologically the condition is explained by supposing that there is a benumbing of the association fibres which normally connect sensory cortical centres with other parts and enable one to compare and adjust new experiences with old—in other words, to reason and form correct judgments. Her mental life is mainly in the sensory-motor centres rather than in the "association centres" of Flechsig. It is a return to the unripe brain of childhood.

Underlying the hysterical mental state there is a condition of *suggestibility*, by reason of which ideas and impressions easily become fixed and dominate the mind. The person becomes self-hypnotized and believes he or she is suffering from things which have no objective existence. The pains, palsies, and anæsthesias of hysteria major are pseudo-delusions, differing from delusions of the insane in that the false belief or idea is a subconscious one.

Trophic disorders. In hysterical paralysis a slight amount of atrophy occurs, but only such as would naturally follow disuse of the part. A very few cases have been reported in which a true neurotic atrophy resembling the atrophy that occurs in neuritis or progressive muscular atrophy was present, and it is even affirmed that degenerative reactions may be elicited. Cutaneous eruptions and dystrophies practically do not exist, or, if present, are the result of complicating disorders.

Visceral symptoms. Hysterical patients often suffer from dyspepsia and constipation, also from anorexia and in some cases from persistent vomiting or regurgitation of food. Occasionally the anorexia and vomiting become persistent; the patient refuses food or rejects all that is taken; she emaciates, becomes weak and bedridden, and develops into that particular phase of hysteria known as "the fasting girl." In these cases, along with the aversion to food and vomiting, there may be a great deal of gastralgia. The urine in hysteria is apt to be of low specific gravity. Always after hysterical attacks there is a profuse flow of very light-colored urine having a gravity of only 1.003 to 1.006. Sometimes there is retention of urine; in extremely rare cases there is a condition known as ischuria and anuria, in which for several days extremely small quantities of urine are passed, owing apparently to a suspension of the functions of the kidney. Such cases should always be carefully investigated, to see that the patient does not deceive her attendants in regard to the amount of urine passed.

Vasomotor symptoms are very common. They consist of flushings and pallor, cold extremities, and at times an œdematous condition of one or more extremities. This œdema may be of the ordinary pale, waxy character, pitting upon pressure. In other cases it has a peculiar bluish tinge and it does not pit; the hands, which are the parts generally affected, are several degrees below the normal in temperature, and the limb resembles in some respects the condition in Raynaud's disease. Gangrene, however, never supervenes. This form of œdema is known as the *blue œdema of hysteria*.

There occur in hysteria febrile attacks, and much has been written upon the subject of *hysterical fever*. These so-called hysterical pyrexias may resemble in their course typhoid or malarial fever; as a rule, the temperature runs a very irregular course, and the fever often lasts for weeks or even months. The essentially neurotic origin of these fevers has hardly yet been established, and one can reach the diagnosis only by most carefully excluding all other possible causes.

Anæmia is a very common condition in hysterical patients.

Hystero-Epilepsy.—The form of hysteria which shows itself by the development of severe crises known as hystero-epileptic attacks is extremely rare in this country, at least in its typical phase. It has been particularly studied by the French writers Charcot, Richer, and others. Hystero-epilepsy, as this form of the disease is called, is a true hysteria and not epilepsy at all, nor a mixture of hysteria and epilepsy, though the name would suggest that that was the case. The typical attacks of hystero-epilepsy begin with certain prodromata consisting of a feeling of malaise and irritability which may last for several hours or a day. The attack is ushered in often with an aura, the patient utters a cry, falls to the ground, loses consciousness, and enters into the first phase, known as the epileptoid stage. During this she suffers from tonic and clonic spasms very much like those of true epilepsy. The muscles finally relax, and the patient becomes comatose for a moment and then enters the second stage, that of the contortions and grand movements. In this there is opisthotonos, the body is arched up, and there are violent movements of the trunk and limbs, which undergo flexion and extension, the movements being all of large range. The next stage is that of emotional attitudes, during which the patient seems to be experiencing intense feelings of anger, joy, or some other violent passion, which she expresses by the postures of her body, the movements of the eyes and facial muscles. This stage over, she enters into the last phase, which is known as that

of delirium, during which there is a great deal of mental excitement of a depressing character, from which she gradually emerges into her normal condition. To recapitulate: we have in a typical attack, first, prodromata; second, the epileptoid phase, lasting from one to three minutes; third, the phase of contortions and grand movements, one to three minutes; fourth, the emotional phase, lasting from five to fifteen minutes; and, finally, the stage

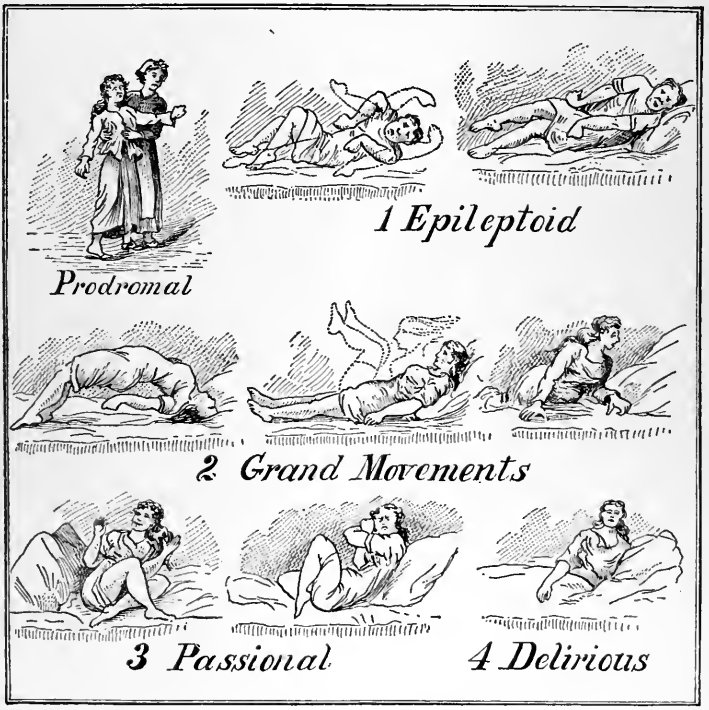


Fig. 225.—Showing the Different Stages of a Hystero-Epileptic Seizure (after Richer).

of delirium, lasting a variable time; the whole attack lasting from five to twenty minutes (Fig. 225). In this country we occasionally see hysterical patients exhibiting one or two of these phases, but very rarely indeed do they ever go through the whole series. The patients who suffer from hystero-epileptic attacks generally during the interparoxysmal stage present many of the stigmata of hysteria, such as paralyses, contractures, and anæsthesias.

Hysterical crises which take the form of convulsions or emotional seizures sometimes end or are associated with attacks of *catalepsy* or *trance* or attacks of amnesia and cerebral automatism.

As these conditions all occur in other diseases than hysteria, they will be described elsewhere in connection with the subject of the disorders of sleep and of consciousness.

Hysterical persons occasionally are attacked with violent and persistent hiccoughing or sneezing. Sometimes also there come on attacks of extremely rapid breathing or hysterical polypnœa, during which the respirations run up to fifty or seventy a minute. A hysterical cough sometimes occurs; it lasts for a long time. Œsophageal spasm with consequent dysphagia is another one of the somewhat rare phenomena of hysteria.

Pathology.—There is no known anatomical change at the basis of hysteria. We do not find the marks of degeneration as we do in certain forms of insanity and epilepsy.

Diagnosis.—Physicians recognize three different phases of hysteria—a hysterical temperament, hysteria minor, and hysteria major. The hysterical temperament is something with which all women and many men are naturally endowed. It is a condition, not a disease, and does not call for description or elucidation here. Hysteria minor is the hysterical temperament plus certain stigmata and the crises. One should not make the diagnosis of hysteria minor unless he can find these factors. The stigmata we have already enumerated, also the peculiar and varied forms in which the crises show themselves. In hysteria major we have a much greater preponderance of the stigmata and much severer forms of the crises, these being largely of a motor type. Hysteria simulates many organic diseases, and it is often difficult to distinguish surely the real from the spurious thing. The essential characteristics of hysterical forms of disease are the peculiar emotional condition of the patient, the past history of hysterical crises, the presence of the stigmata of hysteria such as anæsthesias, limitation of the visual field, paralyses, and contractures. The variability of the symptoms, their susceptibility to influence under suggestion and rigorous moral measures, the absence of serious disturbance of nutrition, the sex and age, and the cause should also have weight in guiding us to our decision.

Diagnosis of special forms of hysterical manifestations. Hysterical paralysis is characterized by the fact that there is no marked degree of wasting of the muscles, no electrical reactions of degeneration, the deep reflexes are preserved or exaggerated, and other marks of hysteria are present. Hysterical anæsthesia can generally be lessened over certain areas by the application of the magnet or can be made temporarily to disappear; it is peculiarly distributed in the way described under symptoms and is associated

with anæsthesias of the special senses. Hysterical contractures sometimes cease during sleep and always under deep narcosis, and the use of an anæsthetic may clear up the case. They usually follow a fit, an injury, or an operation. They are somewhat increased on attempts to overcome them by force; they are usually associated with paralysis and anæsthesia and other hysterical symptoms.

Hysterical convulsions. These differ from convulsions of epilepsy in the way best indicated by the following table:

Hysterical Convulsion.	*Epileptic Convulsion.*
Brought on by emotion or injury; no aura; no initial cry; movements co-ordinate; tongue not bitten, and patient never injures herself. Duration perhaps several hours with intermissions; consciousness generally preserved. Micturition and defecation do not occur. No rise of temperature; may be stopped artificially.	The opposite in all these particulars.

The hystero-epileptic attacks are so characteristic that a mistake could not be made.

Prognosis.—The prognosis of hysteria in children is good. They generally get well, though in some cases there is a recurrence later in life. In hysteria minor of young adults the prognosis varies with the severity of the disease and with the physical strength, mental endowment, and social environment of the patient. Mild forms of hysteria under proper treatment usually get well. The severer forms are often intractable even under the best treatment. When a severe form of hysteria occurs in a person of feeble frame who is surrounded with a sympathetic family, the task of rescuing her from her disorder is a very arduous one. Traumatic forms of hysteria which are not infrequently associated with some actual physical injury are sometimes difficult to cure. Hysteria which is associated with some organic disease, such as a severe pelvic disorder or an organic affection of the central nervous system, has a bad prognosis. Hysteria in the male is generally curable, but it requires vigorous treatment, and spontaneous cure is by no means likely to happen.

Treatment.—The treatment of hysteria may be divided into the mental, mechanical, dietetic, and medicinal.

By all odds the most important factor in the treatment of hysteria is the mental treatment, and the most important measure to

be taken is the isolation of the patient. She should be placed where she will not be surrounded by sympathetic friends; where her life will be a regular one; where some occupation may be given which will engross her attention, interest her mind, and call into play her physical activities. In the major forms of hysteria associated with anorexia, emaciation, anæmia, and possibly pelvic disorders, the "rest cure" as elaborated and carried out by Weir Mitchell forms by all odds the most successful means of treatment. In many cases of less severe character a partial rest cure in which the patient is separated from her family but is not placed under such severe restrictions may be all that is needed. In the case of children removal from home is often advisable, and the discipline of well-conducted schools is a most excellent measure.

The mechanical means used in hysteria are hydrotherapy, electricity, massage, and exercise. Of these measures hydrotheraphy and electricity take the first rank. In hydrotherapy the douche or jet to the back, the shower and cold plunge, and the half-bath are the most efficacious. The technique of their use is given elsewhere. In the electrical treatment the static and faradic currents give the best results. The static sparks often relieve contractures and lessen or remove the anæsthesias, and both forms of electricity seem to have a generally beneficial tonic effect. Massage is of some value in promoting nutrition and it also has a favorable sedative effect on many cases. Exercise, particularly of an active kind such as stimulates the mind and interests one, is a measure of extreme value and one which has perhaps not been sufficiently appreciated. The use of the bicycle, playing tennis, and horseback-riding are measures which cannot be too strongly recommended to hysterical women; in fact, it is probable that some cases which are submitted to the rest-cure treatment might do better by an entirely opposite kind of procedure.

The drugs which can be recommended in hysteria are not numerous and their power is limited. Valerianate of zinc, turpentine, asafœtida, tincture of sumbul, iron, and the bromides are the most important of the nervines. In hysterical children a capsule containing two grains of valerianate of zinc and one of sulphate of quinine is often efficacious. Gowers places more reliance upon the oil of turpentine in doses which should be increased to the point of strangury. Pitres recommends the wearing of colored glasses in order to keep off hysterical attacks. Some experimentation is necessary in order to see which color is most suited to the case.

In the treatment of hysterical convulsions the most efficient measure is the administration of an emetic, and this can be best

done by giving hypodermically one-twelfth of a grain of apomorphine. Convulsions can be stopped sometimes by throwing water in the face or on the epigastrium; by firm and somewhat long-continued pressure over the ovaries; by the administration of valerian, aromatic spirits of ammonia, or compound spirits of ether.

The Spasmodic Tics (Tic Convulsif).

Spasmodic tic is a disease to which the name of chorea is often, but incorrectly, given. It is a very chronic disorder, and shows itself in the form of quick, electric-like spasms of certain groups of muscles or single muscles. The spasmodic movements are violent, and several rapid contractions succeed each other, after which there is a period of rest. The spasm has a tendency to become localized in certain nerves, especially the facial (*mimic tic*), or even in a single branch or twig, as that to the orbicularis, the zygomaticus, the diaphragm, or the tensor tympani. Spasmodic tic sometimes involves the muscles of expiration and the larynx, and then it has been wrongly called *chorea of the larynx*. Stuttering is a form of *tic*.

The convulsive movements may take a wide range and affect a number of groups of muscles, producing quick, violent movements of the body. They are sometimes accompanied by explosive disturbances of speech. In these cases the patient at the time of the convulsive movement utters some obscene or profane words (*coprolalia*), or involuntarily repeats the last words of the sentence spoken to him (*echolalia*), or spasmodically imitates a gesture made to him (*echokinesis*), or involuntarily exclaims the thought uppermost in his mind, perhaps revealing some secret against his will (*tic de pensée*).

The peculiar disorder of the Maine "jumpers," characterized by sudden violent movements on being touched or startled, is a form of tic. So also are the similar troubles known as *latah*, occurring in Malay, and *myriachit*, occurring in Siberia and Kamchatka.

Most of the special forms of spasmodic tic (mimic tic, wryneck, etc.) have been described elsewhere.

Spasmodic tic with coprolalia affects children between the ages of six and sixteen years, and by preference the masculine sex. There is almost always a neurotic family history, and the children are nervous.

The disease begins with attacks of violent and irregular movements, affecting generally the head, face, and upper extremities first, then involving the whole body. The movements can be con-

trolled for a time by the will, only to break out with increased violence later. They cease entirely during sleep, which is generally profound.

After having suffered from the disease for a time, the patient will, with the attacks, utter inarticulate cries, or he may begin to repeat or echo the words that he overhears. All this is done automatically and suddenly, with the accompaniment of grimaces and muscular contortions. The special peculiarity of the disease is the sudden interjection by the patient of obscene words and expressions (coprolalia).

The disorder is chronic, lasting for years. It is best treated by isolation, tonics, and ordinary antispasmodics.

Thomsen's Disease (Myotonia Congenita).

This is a hereditary family disease characterized by the development of tonic cramps when the patient attempts voluntary movements. The disorder is very rare.

Etiology.—Congenital myotony is practically always hereditary and runs in families. It affects males by preference and develops at the time of adolescence.

Symptoms.—The patient notices that on trying to rise or walk his legs are seized with a painless cramp, which in a few seconds relaxes, but comes on again when the muscular movements have been repeated. If he closes his hands tightly a cramp occurs and he cannot relax the grip. If he shuts his eyes he cannot open them for a moment. The muscles of mastication may be affected, but the extremities are the parts most involved. The involuntary muscles are spared. The cramps are increased by cold and nervousness; they are lessened by muscular exercise. The muscles are somewhat hypertrophied, and the patient may present the appearance of a very strong man. The actual strength is fair, but less than would seem. The general health may be good, but the patients sometimes show the signs of low vitality in weak digestion, feeble sexual power, and susceptibility to cold.

The electrical excitability of the nerves is normal, that of the muscles is increased, and there is produced a contraction tetanus by both currents. In addition Erb describes a peculiar reaction produced by a strong stabile galvanic current. It consists in the appearance of wave-like muscular movements passing from cathode to anode. This was not present in my case or Jacoby's. The mechanical excitability of the muscles is also increased.

Pathology.—The disease is probably a primary muscular dystrophy. There may be, however, a peculiar defect in innervation, resulting from a congenital anomaly of the motor tracts. The muscular fibres are found to be hypertrophied, the striations indistinct, and the nuclei increased.

The *diagnosis* is easily made by the characteristic tonic cramps.

The *prognosis* is bad as regards cure, but the disorder does not shorten life.

Treatment.—Dr. Thomsen, who first described the disease, states that active muscular exercise benefits patients. No specific measures are known.

Congenital Paramyotonia.

Paramyotonia is the name given to a form of myotonia in which the symptoms deviate somewhat from the typical ones that appear in Thomsen's disease. Paramyotonia occurs symptomatically, congenitally, and in a peculiar clinical form known as ataxic. We have, therefore, symptomatic, congenital, and ataxic forms.

Symptomatic paramyotonia is noted most characteristically in a certain form of paralysis agitans. Here the patient, when attempting to walk or to rise from the sitting posture, is suddenly seized with an apparent rigidity of the muscles which prevents him from stirring. The myotonic condition appears also in spastic paralyses of spinal and cerebral origin.

Congenital paramyotonia is a family affection, resembling in this respect Thomsen's disease. The muscular rigidity is brought on not by voluntary movements, but by exposure to cold and often very slight degrees of cold. The tonic spasm is a long one and lasts for from a quarter of an hour to several hours. It affects the arms more than the legs. The facial muscles are prone to become rigid. The attacks are followed by some muscular weakness. In congenital paramyotonia the trouble is undoubtedly a primary disturbance of the muscles; in other words, a myopathy.

Ataxic paramyotonia is the name given to a disorder characterized by transient spasms like those of Thomsen's disease, associated with distinct ataxia and also with weakness and some anæsthesia (Gowers). This disease is probably located in the spinal cord and should perhaps be considered one of the forms of symptomatic paramyotonia. No special treatment can be given for either of the two latter forms of disease, of which very few examples have been observed.

Akinesia Algera (Pain Palsy).

Akinesia algera is the name given by Moebius to a peculiar form of paralysis which occurs in psychopathic persons and is due to the fact that intense pains are produced by every muscular movement. The result is that the patient lies helplessly in bed, afraid to stir hand or foot. The disease occurs only in persons who have a very unstable nervous system and generally in those who have a paranoiac tendency. It occurs in adults only. Pain paralysis comes on gradually and affects eventually all the muscles of the extremities and body. There are no objective disturbances such as atrophy, electrical degenerations, and anæsthesias. The muscles and skin, however, are somewhat tender to the touch. The disease

lasts a long time. The patient sometimes improves, in other cases insanity ensues. The disease is essentially a form of insanity, a pathophobia, and is allied to the disorder known as mysophobia. The paralysis is the result of pain hallucination, and the patient is afraid to move the arm or leg on acccount of this hallucination, just as the mysophobic patient is afraid to touch anything on account of the fear of contamination.

The prognosis is bad, and so far treatment has accomplished little or nothing.

CHAPTER XXIV.

THE ACQUIRED NEUROSES.

Chorea (St. Vitus' Dance).

Under this name various spasmodic disorders have been described. They are to be classed as follows:

I. Common chorea, or Sydenham's chorea.

II. Hereditary chorea, or Huntington's chorea.

III. The convulsive tics.

IV. Hysterical chorea, including so-called chorea major.

V. Various local endemic choreas, such as the electric chorea of Dubini and the electric chorea of Bergeron.

Chorea of Sydenham.

This is the common type of chorea, and is the disease ordinarily meant when the term chorea is used. It is a subacute disorder characterized by irregular jerking and inco-ordinate movements. The disease is a common one, forming about one-fifth of the nervous diseases of children.

Etiology.—Most cases occur between the ages of five and fifteen (see chart, p. 476). It is very rare under five. A few cases occur after twenty, and even up to old age, when a senile chorea is sometimes observed. It affects girls more than boys in the ratio of about 2.5 to 1. In adult life the disproportion is less marked.

It is relatively rare in the negro race, especially in those of pure blood (Mitchell). In this country it is more common in children of German, Hebrew, and Portuguese races.

It occurs in all climates. Most cases develop in the spring months, next in the autumn, next in winter, and last in summer. The seasonal influence varies in different localities. In Philadelphia more cases occur relatively in the spring. In New York there is an almost equal increase in the autumn. School attendance has something to do with these variations. Choreic attacks appear to be related to increase in storms (Lewis). The disease is more frequent in cities, and probably in the poorer classes. Hereditary influence is slight, but it exists. In a small percentage of cases one

parent has had chorea, epilepsy, insanity, or a decided neuropathic constitution. A phthisical or a gouty history in parents is also not rare.

The chief exciting causes are injury and fright, mental worry, and rheumatism. Fright or some emotional disturbance is a cause in about one-fifth of the cases. Acute rheumatism is given as a cause in very varying proportions, ranging from five to twenty-five per cent. In this country it ranges from fifteen to twenty per cent (Sinkler, Starr, Sachs, and personal observations). Endocarditis is developed in the course of chorea in a slightly larger proportion of cases. This may exist without any manifestations of rheumatism. Pregnancy is a cause of chorea generally in primiparæ and always in young women under twenty-five. Chorea sometimes follows infectious fevers, especially measles, scarlatina, and whooping-cough. It has been caused in rare cases by reflex irritation from an injury, from nasal disease, and from sexual disorders. Overstudy and the worry of examinations are factors in causing chorea in predisposed and badly nourished children. Intestinal irritations, such as worms, may excite chorea. Malaria also may aggravate, if it does not produce it. Hamilton describes a form of chorea caused by tobacco-poisoning. Anæmia and malnutrition underlie most cases.

Symptoms.—The disease may begin suddenly, but usually it develops slowly, and it is not till one or two weeks that the symptoms are decidedly prominent. It usually begins with irregular twitching of the hand or face on one side. The child winks, grimaces, and drops things from its hand. The foot and leg become affected later and the child stumbles in walking. In two or three weeks the opposite side is involved, but usually less than the one originally affected. In three or four weeks the disease reaches its height. The patient's movements are then almost continuous. The hands can hardly be used and the child has to be fed and dressed; even walking is awkward and difficult. Speech is indistinct and confused from the irregular movements of the lips and tongue. The muscles of respiration may be involved so that the rhythm is uneven. It is asserted that the heart's action is affected also; but this is unlikely.

The choreic movements usually occur both when the muscles are at rest and during volitional acts. In some cases the disease is chiefly characterized by inco-ordinate movements when purposeful acts are attempted. In other cases voluntary movements can be readily performed, and the muscles twitch only when the limbs are at rest. The movements cease, as a rule, during sleep. But the child sometimes sleeps badly on account of the movements. In severe cases attacks of mental excitement and even delirium come on

for several successive nights, and this may be so marked a feature as to form what is called *chorea insaniens*, or *maniacal chorea*. Apart from such phenomena, the mind in chorea is usually dulled, the temper irritable, and the child much harder to manage.

The appetite is poor and capricious, the tongue coated, and the bowels are often constipated. The nutrition fails a little; there are anæmia and a tendency to loss of flesh.

The eyes present nothing abnormal. Hypermetropia, astigmatism, and muscular insufficiencies exist, but not much more than in other nervous children.

The child is often worse in the morning and improves toward night. Excitement and physical exertion make the movements worse. There is rarely any pain and never anæsthesia or tenderness. The muscles are weak but not actually paralyzed. The deep reflexes are somewhat lessened and the knee jerk may be abolished. The electrical irritability of the muscles is, as a rule, increased, but there are no qualitative changes. Nocturnal enuresis occasionally occurs. The urine contains an excess of urea and phosphates, and at the height of the attack the specific gravity may be increased.

Forms.—Maniacal chorea is characterized by great mental excitement—especially at night, delirium, with hallucinations and delusions. After one or two weeks the excitement lessens and the patient becomes dull and apathetic. Such cases usually occur in adult women, and they are sometimes fatal.

Paralytic chorea. In this form one arm becomes rather suddenly weak and powerless. A few twitching movements are observed. This form occurs only in children and runs the same course as the spasmodic type.

Chorea of adult life and senile chorea. The disease when it occurs in the second half of life attacks men rather oftener than women; it is not related to rheumatism. There is usually a neurotic family history and even a hereditary history of chorea. The attack is usually caused by emotional disturbances. It runs much the same course as juvenile chorea, but it rather more apt to become chronic. When it occurs in old men it is called *senile chorea*. This type is not to be confounded with hereditary or Huntington's chorea.

Duration—Relapses.—The disease in this country lasts about ten or twelve weeks, ranging, however, from six weeks to six months. There may be great improvement followed by a relapse, and in this remittent manner the disease may last for years. If it last more than six months it should be called *chronic*. Relapses occur in about one-third of the cases and rather oftener in girls. Relapses occur oftenest within a year of the first attack and much

oftener in the spring. After three years relapses practically cease. The number of relapses is usually but one, but the disease may recur eight or nine times. Relapses rarely occur in adults except in the chorea of pregnancy.

Pathology.—The seat of the lesions in chorea is the gray matter of the cortex and its meninges, the pyramidal tract, basal ganglia, and the spinal cord. The lesions are in acute cases of the

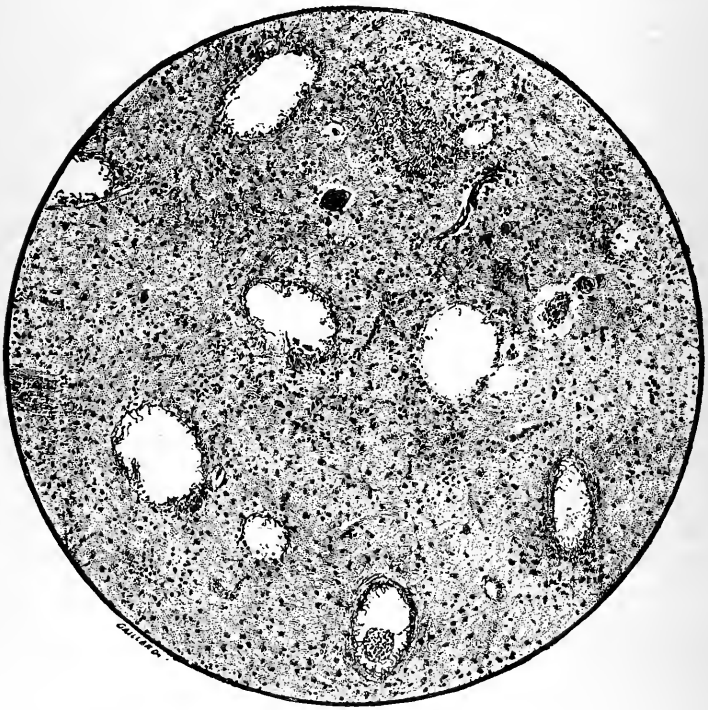


Fig. 226.—Perivascular Dilatations in the White Matter of the Convolutions of a Very Chronic and Severe Case of Chorea.

nature of intense hyperæmia, with dilatation of vessels, small hemorrhages, and spots of softening. There are infiltration of the perivascular spaces with round cells and swelling and proliferation of the intima of the small arteries. In chronic cases the evidence of active vascular irritation is less, but there are perivascular dilatations and increase of connective tissue (Fig. 226). The process suggests a low grade or an initial stage of inflammation. The cause of this is probably either an infective micro-organism or a humoral irritation similar to that causing the rheumatic symptoms

and the heart lesions. In a considerable per cent of cases (ninety per cent—Osler), especially in those of long duration, there are fibrinous deposits on the walls of the heart. The hyperæmic process may not be confined to the meninges and motor areas of the brain and cord, but it is only from the disease in these parts that the symptoms of chorea arise. The presence of points of irritation in the cortex and its meninges and in the deeper parts excites irregular discharges of nerve force and produces the choreic movements. The interruption of the voluntary nerve impulses by diseased foci makes these movements irregular. The apparently special involvement of the lenticular nuclei may explain some of the inco-ordination. In paralytic chorea the pyramidal tract is probably more seriously injured by some single large focus of congestion, exudation, or hemorrhage. Indeed, I have seen a true hemiplegia develop in the midst of an attack. In maniacal chorea the meninges and cortex are more involved.

Diagnosis.—The disease is easily recognized by the peculiar twitching movements. It is necessary only to distinguish the different forms. It must be distinguished from convulsive tic, electric chorea of Dubini, hysterical spasms which include myoclonus, saltatory chorea, and chorea major. The distinctions are not difficult and are given in connection with the descriptions of these disorders.

Prognosis.—As regards life the prognosis is very favorable. In this country death from chorea hardly ever occurs in children. It is more fatal in adults. In England the mortality from chorea is about two per cent. Nearly all non-fatal cases eventually get well.

Treatment.—The most important single factor in treatment is rest. The child should not be allowed to take violent exercise or to have any excitement. In most cases he should be taken from school, and in bad cases he should be kept in bed.

Cold sponging or the ether spray daily along the back is useful. Nourishing food and iron are indicated.

As specific remedies, arsenic still heads the list. It should be given in doses of ℥v. of Fowler's solution t.i.d., increased by one or two drops daily to fifteen or twenty drops or even more. If this causes nausea and gastric pain or headache, the dose should be stopped for a day and then resumed, if possible, where it was left off. Sometimes the sulphide of arsenic is better tolerated than the arsenite. Next to arsenic come antipyrin (gr. v.), antifebrin (gr. iij.), exalgin (gr. iij.), the doses to be carefully increased if needed. Tincture of cimicifuga sometimes helps when arsenic fails. The bromide or valerianate of zinc is also an excellent remedy, especially when there is a hysterical element. The bromides and chloral are

useful adjuvants in promoting sleep. Chloral alone is said to be curative if given in doses sufficient to prolong sleep greatly (Bastian). Exalgin given cautiously with iron, in doses increased to fifteen grains a day, is often very useful. Hyoscine hydrobromate in doses of gr. $\frac{1}{100}$ is occasionally efficacious. Among other drugs of less value are the salicylates, cypropedium, lobeline, physostigmine, and tartar emetic. In chronic and obstinate cases hypodermic injections of Fowler's solution should be tried. Galvanization of the brain and spine is also useful. Change of air sometimes breaks up an attack.

Hereditary chorea was first described by a Long Island physician, Dr. Waters, in 1842, later by Drs. Gorman and Lyon, and in 1872 by Dr. Huntington. The American cases have been observed chiefly in New York, Connecticut, New Jersey, and Pennsylvania. Cases have been reported also from Germany, France, and England. The disease rarely begins before thirty or after fifty; it occurs about equally in males and females. It is always directly hereditary, either through father or mother, usually the latter. It begins without known cause by twitchings in the face; the movements then extend to the arms and legs. It is attended by progressive mental deterioration, by a tendency to melancholia, and finally ends in dementia. Its course is chronic and usually very slow, lasting ten or twenty years. Post mortem, chronic pachymeningitis and leptomeningitis with degenerative changes in the cortex have been found.

Electric Chorea is a name sometimes and wrongly given to very violent forms of ordinary chorea of Sydenham. The term was first applied by Dubini to a peculiar and progressively fatal spasmodic affection which has been observed almost solely in Italy, and which is perhaps of a podagrous or malignant malarial origin. M. Bergeron in 1880 also described an "electric chorea" in which the patients are attacked by sudden rhythmical spasms. This latter disease has a uniformly favorable course. Neither of these diseases resembles true chorea, nor do they have the character of the *tics*.

The term electric chorea, therefore, is one that should be used, if at all, only with a qualifying explanation.

Habit Chorea (*Tic Coordiné*).—There are many persons who go through life with some trick of speech, of gesture, or some peculiar grimace. It may be only a shrug of the shoulder, a twitching of the eyes, or a sniff. These various movements are *tics* of the co-ordinate kind. The spasmodic motion is of itself normal, but is inappropriate and misapplied.

Such movements are often seen in children. They sometimes represent abortive attacks of chorea, and sometimes they are the residuum of old attacks. In many cases they are chronic convul-

sives tics from the start and have little relationship to Sydenham's chorea. The condition is to be treated both by moral and medicinal measures.

Oscillatory spasms and nodding spasms have been described elsewhere.

Procursive Chorea, or Dancing Chorea.—Laycock has described as a separate kind of chorea a rhythmical or trochaic form, which he says affects children, principally girls, and shows itself in spasmodic rhythmical contractions or in sudden rotating or procursive movements of the body. This has been called chorea procursive, or chorea festinans, by other writers. In many cases it is accompanied by vertigo, when the condition of the patient is similar to that of a person who has been whirling around a number of times. Such cases always have decidedly hysterical characters, although these procursive attacks may complicate ordinary chorea.

Chorea major is a manifestation of hysteria, and has been described under that head. It is not a chorea at all.

Paramyoclonus Multiplex (Myoclonus Multiplex, Convulsive Tremor, Myospasia).

Myoclonus multiplex is a rare disease allied in nature to the convulsive tics and characterized by attacks of quick clonic spasms affecting the trunk and sometimes the extremities also. The disease occurs most often in adult males. It is caused usually by fright, injury, or some violent emotion. A condition resembling it is produced by removal of the thyroid. Very different forms of spasm have been described under the name myoclonus multiplex, but there are two groups of cases which may be distinguished. One is of hysterical type, the other belongs to the convulsive tics, and in its typical form myoclonus is to be classed with this latter form of spasmodic disorder. In the hysterical form the patient without warning is seized with sudden and lightning-like contractions of the trunk and hip muscles, which cause his body to be alternately flexed and extended so violently that he is often thrown from the chair or couch on which he is lying. The arms and legs may be also involved, and the attack takes on the characteristics of a general tremor in which the whole body shakes. The attack then ceases and the patient has a rest for hours or days. In the true or choreiform type the disease comes on more slowly, with sharp choreic-like twitchings, and the patient presents something of the aspect of chronic chorea. The facial muscles may be affected, but not those of the eyes. The spasmodic movements are bilateral, although they sometimes begin on one side. The convulsions differ from those of hysteria in that they affect groups of muscles that have not the same physiological function and produce movements which cannot be easily imitated voluntarily. Still this distinction is not a sharp one. The patients are generally neurasthenic. They

have no paralyses, no anæsthesias, very few pains, and their bodily nutrition is not seriously impaired.

The pathology and pathological anatomy are unknown.

The *diagnosis* is based on the peculiar character of the spasm, on the fact that the trunk muscles are involved, and that the spasms are bilateral. The disease usually lasts but a few months in the hysterical form, but it may extend over a number of years.

The *prognosis* is fairly good.

The *treatment* consists of tonics and the use of chloral and hyoscine. Galvanism seems to be very efficient. Atropine and hyoscine are indicated, and hydrotherapy may also be advantageously employed. Thyroid extract has been of benefit.

Saltatory Spasm.

This is a curious and rare form of disease characterized by convulsive movements of the legs brought out by touching the feet to the floor. It occurs in both sexes and at all ages, but usually in those of a neurasthenic and hysterical temperament. The phenomena of the disease are exhibited when the patient attempts to stand. The minute that the feet touch the floor violent contractions occur in the muscles of the calves and hips, sometimes in the whole body; these cause the patient to jump, and the movements may be so severe as to throw him down. The seizures are brought on only by the exciting effects of the weight of the body on the feet. Saltatory spasm is probably a form of hysterical spasm. It has been described chiefly by the older writers, and its symptomatology and pathology have not been carefully worked out.

Tetanus.

Tetanus is an acute or subacute infectious disease characterized by violent tonic spasms with remissions and exacerbations. It is called idiopathic when no open wound is found and traumatic when such condition is present. When it attacks infants it is called tetanus neonatorum; when the jaws alone are involved it is called lockjaw, or trismus. A form which affects the face and throat is called head or cephalic tetanus.

Etiology.—It has a special predilection for newborn children in some countries (West Indies) and to a less extent for puerperal women. It affect males more than females. After the first month of life there is practical immunity till after the tenth year. It then increases in frequency up to forty. It is much more frequent in dark races and in some tropical climates (West Indies, South and Central America).

Symptoms.—The disease sets in from five to fifteen days after infection. It begins with feelings of stiffness in the neck and throat and sometimes with chilly feelings. Gradually tonic spasms develop which involve the trunk muscles, causing opisthotonos and other forms of rigid spasm. Trismus, or lockjaw, also occurs. The spasms are attended with intense pain. Sometimes there is a rise of temperature and this may be very high. The disease lasts from two to five weeks. There is evidence of irritation and congestion of the spinal cord and injured nerves, but no special anatomical changes are found. A specific bacillus producing a tetanizing poison has been discovered.

The *diagnosis* is based on the characteristic history and the peculiar spasms. In strychnine posioning there is no initial trismus or epigastric pain. In rabies there is also no trismus but a respiratory spasm on attempts to swallow.

The *prognosis* is bad. About eighty per cent of traumatic and sixty per cent of idiopathic cases die.

The *treatment* consists of complete rest and quiet in a dark room and the administration of chloral, bromide, morphine, and physostigma. Successful results from injection of blood serum of an animal which has had the disease are reported. A tetanus antitoxin has also been tried with some success, and this has even been injected into the subdural cavity of the brain, but the results are not yet very satisfactory.

Tetany (Tetanilla).

Tetany is a subacute or chronic spasmodic disorder characterized by intermittent or persistent tonic contractions beginning in the extremities and associated with paræsthesiæ and hyperexcitability of the motor and sensory nerves.

Etiology.—The disease is very rare in this country, but relatively common in Europe, especially in Austria. It occurs with frequency during the second, third, and fourth years of life and again at the time of puberty. Its rate of frequency then slowly declines and it is very rare after fifty. It affects males much oftener than females up to the age of twenty; after that the difference disappears. It occurs mostly in the working classes. In infants rickets is often noted. The exciting causes are exhausting influences like diarrhœa, lactation, sepsis, fatigue, mental shock, and fevers; also exposure to cold and wet. Alcoholism, dilatation of the stomach, and intestinal entozoa are also causes. It may be produced artificially by extirpation of the thyroid gland. The disease sometimes appears as an epidemic.

Symptoms.—Tetany begins sometimes suddenly with symmetrical

tonic contractions of the hands; at other times there are at first sensations of numbness, prickling or pain in the extremities, with malaise and perhaps nausea; then spasms begin. The attacks affect first and most the upper extremities. The flexors of the forearm and hand are usually involved; the fingers are flexed at the metacarpo-phalangeal joint and extended at the other joints, and the thumb is adducted, producing the "accoucheur's hand." The forearm may be flexed and the upper arm adducted. The knees and feet are extended, the toes flexed, and the foot is inverted. In severe cases the muscles of the abdomen, chest, neck, and face are involved. Opisthotonos and dyspnœa may result. The muscles of the face and eyes develop contractions, and trismus sometimes occurs late in the disease. The muscles of the larynx, œsophagus, and bladder may be affected. Fibrillary tremors are observed in the contracted muscles. The attacks are accompanied by paræsthesias and cramp-like pains. There may be some abolition of sensation in the skin of the parts affected during attacks. The cramps last from a few minutes to hours or days. They occur during day and night and may wake the patient from sleep. Fever is sometimes present in epidemic cases. The disease has a tendency to recurrence.

While it lasts, both during and between the attacks peculiar phenomena are observed as follows:

1st. *Increased Mechanical Irritability of Motor Nerves.*—The motor nerves show an abnormal irritability, so that on striking the motor point a sharp muscular contraction is brought out. When pressure or a blow is made on the face over or near the exit of the facial nerve from its foramen, contractions of the facial muscles occur, especially those of the lips. This is called the "facial phenomenon." By pressing on the artery and nerve of a limb a tetanic attack can be produced in the muscles supplied. It is probable that it is the pressure on the nerve alone which causes the phenomenon which is called "Trousseau's symptom."

2d. *The electrical irritability* of the muscles and nerves is increased, especially to the galvanic current. Thus a negative-pole closure contraction (CaCC) is brought out by a very weak current; and if a little stronger it causes a tonic contraction or cathode-closure tetanus (CaCTe). The positive-pole opening contraction (AnOC) may be tetanic, *i.e.*, AnOTe, and there may be even a cathode-opening tetanus (CaOTe), a phenomenon not seen in any other disease. According to Gowers there may be a reversal of the polar formula, so that a positive-pole closure contraction occurs earlier than a negative (AnCC > CaCC). This is certainly rare.

3d. *An increase of irritability of the sensory nerves* is shown by pressing upon them, when sensations of prickling and formication appear along their course. There is an increase also in the electrical sensibility, shown by appreciation of very weak galvanic currents. The auditory nerve reacts to the galvanic current in about fifteen per cent of normal cases, and then only to strong currents

and to only a partial extent; but in tetany it reacts in nearly all cases, and with comparatively weak currents (2 to 5 or 6 ma.) on anode closure, anode fixed, and anode opening (AnC Klang, AnDKl, AnOKl) (Chvostek).

The phenomena of hyperexcitability above described vary considerably and rapidly during the course of the disease, and are not always present.

Types of the Disease.—The disease varies in intensity and duration. This variation depends much upon the cause, and there have been made a number of types of the disease based on the etiology. Thus we have:

1. Epidemic or rheumatic tetany.
2. Asthenic tetany due to lactation, diarrhœa, exhausting diseases, etc.
3. Thyroid tetany, due to removal of the thyroid gland.
4. Reflex tetany from gastric dilatation and intestinal worms.
5. Latent forms of tetany in which the phenomena of hyperexcitability and paræsthesia occur with very slight if any contractions, and no Trousseau symptom.

Infantile tetany should perhaps be separated from other forms.

Symptomatic tetany from brain disease is also spoken of.

When the spasms are continuous the disease lasts but a few weeks; when they are intermittent it may continue for months. Epidemic cases last but a few weeks. The disease may be said in general to last from a few weeks to a few months. Patients are liable to a recurrence on return of the exciting cause.

Pathology.—The phenomena of the disease indicate a congested and irritative condition of the gray matter of the spinal cord. The cause of this state is evidently in some cases (epidemic tetany) an infectious poison; in other cases mucin in the blood (thyroid tetany), and in other cases it may be a rheumatic or some other toxic influence. Ergot is known to produce symptoms resembling tetany.

In infantile tetany the irritation is apparently cortical and due to meningitis or to rickets and the reflex irritation of disordered bowels. It is doubtful if any reflex influence can be invoked in adults. Tetany is a functional disease and the symptomatic expression of a central irritation. This irritation may be of different kinds, hence tetany has a claim to be called a distinct disease simply on clinical grounds. It has no such definite pathology as chorea or epilepsy. In the very few autopsies which have been made no definite organic lesion has been found.

Diagnosis.—The disease is usually easily recognized by the character of the spasms, their symmetrical nature, their course, and the phenomena of hyperexcitability of the muscles and nerves. Trousseau's symptom is found in no other disease. The "facial phenomenon," the peculiar electrical and mechanical irritability of the muscles and nerves, are very rare in other conditions. The sensory irritability, and especially that of the acoustic nerve, is also characteristic. From tetanus the disease is distinguished by the intermit-

tency of the contractions, their feebler character, the fact that they begin in the extremities and extend to the trunk, and by the absence of trismus, at least until late in the disease.

Treatment.—The cause should be removed if possible, lactation stopped, diarrhœa and indigestion corrected, worms expelled, rickets if present attended to. Rest, nourishing food, and tonics are indicated. Symptomatically, bromide of potassium in doses of ʒ iss. to ʒ ij. daily with chloral furnishes the surest relief. Hyoscine in doses of gr. $\frac{1}{100}$ may be tried. Inhalation of chloroform or injections of morphine are needed in severe cases. In nocturnal tetany Gowers advises digitalis. Lukewarm baths may be of service; so also may ice bags to the spine. If electricity is used only the weak galvanic current should be employed.

CHAPTER XXV.

NEURASTHENIA (NERVOUS EXHAUSTION, BEARD'S DISEASE).

Neurasthenia may be defined as a chronic functional nervous disorder which is characterized by an excessive nervous weakness and nervous irritability, so that the patient is exhausted by slight causes and reacts morbidly to slight irritations.

There are evidences that the neuropathic constitution existed in all ages, but coherent descriptions of clinical types like the neurasthenia of modern days are not found in literature until the present century. The credit of calling attention to this condition most insistently, most acutely, and most successfully is due to Dr. Geo. M. Beard. He showed that a large class of symptoms which had been previously referred to hypochondriacal fancy, to disease of the stomach, disease of the uterus, perverted conditions of the liver and urinary excretions, were really and fundamentally dependent upon a morbid weakness of the nerve centres, and the result of his writings and the propaganda which he started is that now practically all medical men of experience agree that there is a morbid condition of which the underlying cause is a nervous irritation or defect.

Etiology.—Some doubt has been thrown over the question of the excessive nervousness of the civilized nations of the present time. It is not a matter which can be fairly settled by statistics or the perusal of historical documents, but, on the whole, the evidence is, to my mind, conclusive that the human race does now suffer relatively more from nervous irritability and exhaustion, in its various types, than it did in the past. This I infer partly from the fact that the predisposing and exciting causes of neurasthenia are more largely present now than they used to be. The tendency of people to city rather than rural life is perhaps one of the strongest points in favor of this view, since we know it is in our urban population that neurasthenia breeds best. A larger proportion of persons now also use their brains in the struggle for existence and live upon a higher mental plane, with all the danger which that implies. The eighteenth-century writers attributed all the functional disorders then known, under the terms "vapors," "spleen," "hypochondria," "hysteria," to three things—luxurious living, sedentary life, and

the unsanitary conditions of great and populous cities. They said nothing about the effects of overwork, continual anxiety, and mental strain, and one certainly does not gain from reading the English medical literature of this period that there was any such excess of work and worry among the people.

Without going into any further argument upon this point, I shall admit, as an offset to this, the more widespread knowledge of how to live and how to ward off disease, so that it is at least probable that even if neurasthenia is more prevalent now, and has been steadily increasing, this will not necessarily always continue to be the case.

At the present time we know that neurasthenia is found more frequently among the highly cultivated races. I have seen it in negroes, but it is extremely rare, while hysteria and insanity are fairly common. I believe that Americans deserve to an extent the reputation which they have of suffering greatly from neurasthenia. This is particularly the case in the Northern and Central States, on the Colorado plateau, in parts of California, and in the great cities of the East. Neurasthenia is said to be quite prevalent in Russia, and it is generally observed that it affects particularly often the Hebrew race. In this country we see it quite often in the Irish, but almost as often in the English, and rather less frequently in my experience in the Germans. Neurasthenia prevails rather more in dry temperate climates, but it is by no means infrequent in the tropical regions, and is to be found in the West Indies and in the republics of South and Central America in its classical forms. Neurasthenia is found rather more often in men than in women, but the difference is not great.* Among 828 neurasthenics whose histories were analyzed by Hösslin, there were 604 men and 224 women, but this does not give the proper ratios if we include all grades of society.

The neurasthenic age ranges from eighteen to fifty-five, but the larger proportion of cases is met with between the years of twenty and fifty. Occasionally symptoms resembling neurasthenia may be seen in children of the age of twelve or thirteen, and occasionally also there develops a kind of senile neurasthenia, which is, however, often associated with hypochondriasis, and some definite degenerative changes in the nervous or vascular system.

* Among 100 consecutive personal cases there were 53 women, 47 men. Ages: fifteen to twenty-five, 25; twenty-six to thirty-five, 38; thirty-six to forty-five, 25; forty-six to fifty-five, 20; fifty-six to sixty-five, 2. There are relatively more cases in the adolescence of man and in the later period of life of women. Nativity: United States, 59; Ireland, 29; Germany, 12; others, 9.

In men neurasthenia occurs more often in the single; in women the relation is somewhat reversed, so that, taking both classes, the married and the unmarried are about equal.

Neurasthenia does not much affect the people of the country and small towns, though it does exist there. In great cities the number of neurasthenic women, among the wives of laborers and artisans, is rather large, and this is the natural result of the strain of living with husbands who are dissipated, and of rearing large families of children in the close quarters of a tenement house. The disease is relatively more frequent in the educated classes.

Hereditary influence plays a very considerable part in the development of neurasthenia. We can usually find that there is a history of migraine or some nervous irritability upon one side or the other. A distinct history of the major neuroses or of severe mental diseases is rare, but there is no doubt that a very large proportion of neurasthenics come into the world with an oversensitive and weakened nervous system. They may be strong enough to undergo the ordinary strain of life, but break down under some specially exciting cause.

The exciting causes of neurasthenia are very various, but they can most of them be classed under the head of excessive mental strain or shock, sexual abuse, and the influences of exhausting fevers, of chronic infections like syphilis, and of poisoning with alcohol and tobacco or tea and coffee. In the larger proportion of cases of men, the trouble if it develops during adolescence is brought on by overwork at school and in college, combined with neglect of sleep and carelessness in diet. Frequently the abuses of the sexual function, of tobacco, or of athletics are the exciting causes.

The practice of masturbation is one of the things for which neurasthenics very often keenly reproach themselves and over which much hypochondriacal brooding develops. Excesses of this kind, however, are usually a sign of a degenerate or unbalanced nervous system rather than a cause. The actual harm done is greatly exaggerated, however strongly this practice is to be reprobated. Excessive and unnatural indulgences, such as sodomy, etc., tend to weaken the nervous system and are causal factors of neurasthenia. Bad methods of education and in particular excessive study are thought to predispose to nervous exhaustion. This is usually seen in ambitious college students, or in young men who are forcing their way under great disadvantages through professional schools and into professional practice. Young women, who are excessively devoted to study and yet cannot refrain from social indulgences, sometimes break down with nervous exhaustion. The studies and training of

the primary and secondary schools may prepare the way for these catastrophes, but they rarely come before the eighteenth year.

Typical attacks of neurasthenia are undoubtedly brought on by the fright and shock incident to severe injuries or exposure to great danger, as in railroad collisions and other frightful forms of accident. A large proportion of the so-called "traumatic neuroses" are simply forms of neurasthenia. Neurasthenia can be brought on also by excessive child-bearing, the drain of lactation and domestic trouble, great excesses in eating and drinking, and the strain of hard domestic life and of sickness and nursing. Neurasthenia sometimes follows an acute infection like that of typhoid fever or the grippe. It may also be induced by the infection of syphilis. It then comes on in the secondary or less often in the tertiary stage (Fournier). It is probable that in many of these cases the trouble is due to the excessive use of mercury and saline purges. At any rate antisyphilitic treatment can certainly bring on or bring out a neurasthenia. A combination of secondary syphilis with the excessive use of alcohol leads to a very obstinate type of neurasthenia. Malarial poisoning seems also to have some influence as an exciting cause. Much weight has been laid upon the importance of eye strain in producing neurasthenia, and, given a neuropathic constitution, there is no doubt that the defect in the refraction of the eye or in muscular equilibrium may cause, or at least keep up, a neurasthenic state. The same is probably true of severe forms of gastric disturbance, and of disease of the pelvic organs, such as subinvolution, decided displacements, and chronic ovaritis or salpingitis. In men the existence of prostatic irritation, of irritable strictures, and hemorrhoids and fissures, may start up neurasthenic symptoms. Chronic middle-ear disease and nasal stenosis are also put down as occasional exciting causes. The existence in neurasthenics of a tendency to constipation, or what is popularly known as "biliousness," accompanied by a gouty or lithæmic diathesis, has been much dwelt upon, and at one time neurasthenia was thought to be largely the expression of a disturbed state of the metabolism—a phase only of gout or lithæmia. This tendency, however, is rather the result of the weak nerve centres than the cause, though the two often act in a vicious circle. Prolonged and severe dyspeptic disturbances, especially when associated with atony of the stomach and bowels and the condition known as enteroptosis, are exciting or maintaining causes.

I would sum up the leading causes of neurasthenia thus:

1. Hereditary nerve sensitiveness.
2. Overwork and worry.

3. Severe shocks, with or without injury.
4. Infections.
5. Abuse of stimulants and narcotics.
6. Abuse of sexual functions.
7. Disorder of digestive functions and auto-toxæmia.

This means that the causes are most often a bad heredity and foolish living.

A great deal of stress has been laid upon autotoxæmia as a cause of neurasthenia. There is no doubt that a great many symptoms and crises are brought about through this agency, but the attempts to prevent autotoxæmia by perpetually stimulating the liver and giving intestinal antiseptics, of using a large amount of water and the simplest kind of diet, do not of themselves cure the disease, unless measures are taken to strengthen the impaired tone of the general system.

Symptoms.—The symptoms of neurasthenia, while manifold, have yet a pretty distinct general resemblance to each other, and the clinical picture of typical forms of neurasthenia is quite as pronounced as that of other nervous maladies. The patient's symptoms are to be sure nearly always of the subjective character, but they are reiterated with so much force and feeling, and the independent descriptions tally so closely, that one can hardly fail to be convinced by the story itself that they are expressions of the same morbid condition.

The patient complains of a general feeling of mental depression —life is not the interesting spectacle to him that it formerly was. The man who once delighted in work can hardly force himself now to go to it. He tires very quickly over tasks which were formerly easily performed. He loses his power of originating plans and of mapping out work. He absolutely cannot pursue a train of thought or a single line of work for a long time, but sits idly at his desk or goes back to his home in depression and despair. He is very easily irritated at things which before caused him no annoyance, and becomes a source of domestic unrest and unhappiness. He is oppressed with the fear that he will never get well, or is going to become insane or paralyzed, or that some dreadful termination of his present malady is bound to occur. He sleeps badly, waking up perhaps after a short rest in the early part of the night, or, if he sleeps until morning, he has disquieting dreams, and wakes up unrefreshed. He suffers from a number of peculiar sensations which are called "cephalic paræsthesiæ." These are sensations of pressure on the top of the head, or a feeling of constriction around the temples, or a burning spot on the vertex, or tenderness of the scalp. Sometimes

he has a sense of weakness or even pain in the back of the neck. He has also peculiar paræsthesiæ of the hands and limbs; they feel numb or asleep at times. Peculiar chilly sensations creep up the back or legs. He less often has attacks of dizziness; spots come before the eyes and buzzing sounds are heard in the ears and head. Headache occurs in perhaps one-half of the cases, the headache being usually either frontal or occipital. It is often very persistent, and in fact a chronic headache, not due to tumor or meningitis or syphilis, is almost invariably of neurasthenic origin. This neurasthenic headache is usually diurnal only, coming on in the morning when the patient wakes up, and lasting a good part of the day. It does not often keep him awake at night. In this point it is distinguished from the headaches of syphilis and of meningitis or of tumors. Women suffer from these headaches, and from pains in general, more often than men. They in particular have much pain in the back of the neck and along the spine. This keeps them from walking or being upon their feet, and it may develop into a form of neurasthenia known as "spinal irritation."

The special senses are not very seriously affected. The patients can often see quite well, but their eyes soon tire; the effect of watching a play fatigues them. They cannot read a book long because it makes the eyes smart or produces some headache. Examination of the neurasthenic's eye frequently shows the existence of some refractive error, most frequently astigmatism and hypermetropia; defects in the ocular muscles, and especially weakness of the internal recti, often occur. Patients have frequently complained to me of a defect in visual memory. They see a thing or face but do not remember it again as readily as they used to. There is no limitation of the visual field in true neurasthenia uncomplicated by organic disease, but there is a morbid susceptibility to fatigue, particularly of the periphery of the vision, so that, after long testing, objects in the periphery become less distinct, and a sort of artificial limitation of the field may be produced. In some cases an object which is brought from without into and across the visual field is seen in wider range than an object which is placed in the centre of vision and carried gradually out toward the periphery. This is the reverse of the normal condition, and is known as "Foerster's shifting type." Peculiarities of accommodation, a slight drooping of the lids, inequality of the pupils, and excessive mobility of the iris have been noted in neurasthenia.

As I have already stated, neurasthenics sometimes suffer from tinnitus, which is very distressing and aggravates every other nervous symptom, but this usually occurs only in connection with ac-

tual disease of the middle ear, or in old people with degenerative changes in the cerebral blood-vessels. An excessive sensibility to noises, and even the pleasant sounds, like those of music, may be present. Neurasthenics sometimes cannot bear even the most enchanting melodies. A similar morbid sensibility to taste and smell may be present. But these are matters of minor moment, and are much more often seen in hysteria or in a hystero-neurasthenia.

There is no doubt in my mind that in neurasthenics the general muscular and nervous strength is lessened, and although the patient may not have lost flesh, and may not appear particularly weak, he tires quickly on ordinary exertion, and the tests of the dynamometer show a lessened response. A fine tremor of the hands is often present, and when the eyes are tightly closed there will be a quivering of the lids, and in very acute and exaggerated cases twitchings of the muscles of the face and tongue, almost like those in general paresis. This rarely occurs, however, unless the patient has, in addition to the neurasthenia, a considerable amount of toxæmia from alcohol, tobacco, or tea.

The reflexes are exaggerated very greatly. In many cases a blow upon the leg, anywhere from the patella to the middle of the shinbone, will bring out a prompt reaction, and similarly a blow struck on the thigh anywhere from the patella up half-way along the thigh, will produce a knee jerk. And blows upon the motor points promptly bring out responsive contractions. The cutaneous reflexes are also exaggerated. These things vary considerably, however, in different cases, and are more marked in the younger patients and those of a neuropathic constitution.

The sexual function is irritable and weak.

There is a considerable disturbance of the heart function in neurasthenia. The most frequent condition is an acceleration of the pulse beat from very slight cause, due to a weakening of the inhibition of the heart. A pressure over some painful point in the body will sometimes bring up the pulse from 80 or 90 to over 100, and it will remain there for one or two minutes. This is called "Rumpf's symptom." Arrhythmia and palpitation of the heart are less frequently observed. It is my belief that cardiac weakness is an important condition in many forms of neurasthenia and underlies sometimes a good many of the other symptoms. This is particularly true of the neurasthenias of more advanced life. The cardiac disturbances are more frequent in women, in young people, and in neurasthenia associated with the use of tobacco and tea.

A great deal of emphasis has been laid upon the vasomotor disturbances of neurasthenia, and a large number of neurasthenic symp-

toms have been ascribed to a weakening of the vasomotor centre. As a result of this the patient suffers from cold hands and feet, from flushing of the face alternating with pallor, from dermographic skin, and from those symptoms which we usually attribute to cerebral congestion, such as a sense of fulness in the head, headache, spots before the eye, dizziness, and noises in the head. Sphygmograms of the pulse show a lowering of arterial tension, and perhaps still more a great variability in the tension of arteries..

The condition of the urine has been studied very closely in connection with this subject. In fact many of the symptoms which we now call "neurasthenic" were described by Dr. Prout and Dr. Golding Bird early in the century and were held by these gentlemen to be due to oxaluria. This was a condition characterized by flatulent dyspepsia, melancholia, and nervous irritability, and was thought to be due to defective metabolism, resulting in the production of an excess of oxalic acid. More recent studies have shown that oxaluria is only one of the manifestations of lithæmia, and that while it is significant, as was then supposed, of defective nutritive changes, these are more dependent on a neurasthenic state than primarily upon dyspepsia and metabolic disorder. There are, according to Herter, few cases of neurasthenia which do not show in the urine or fæces some indication of defective metabolism. "The fæces often contain excessive amounts of urobilin or some related substance. The urine is usually concentrated and of small volume (600 to 1000 c.c. in twenty-four hours). Frequently there is an excessive excretion of phosphoric acid (P_2O_5) and an alteration in the quantitative relation of urea and uric acid. In health the relation of the uric acid to the urea excreted varies between 1 to 45 and 1 to 60 in adults. In neurasthenia (as well as some other conditions) the relation is often 1 to 40, 1 to 35, or 1 to 30. Indican is often present in pathological quantities, especially in cases of sexual neurasthenia. Oxalate of lime is often present in excess in the urine" ("Diagnosis of Nervous Diseases," p. 547).

In rare cases one finds in neurasthenics a temporary albuminuria; I have observed it only once in one hundred cases, which is about the average. This albuminuria is not associated with the presence of casts or other evidence of kidney disease, and it is apparently due to a paresis of the vasomotor nerves of the kidneys. Transitory glycosuria is more often found. This glycosuria is usually associated with a heavy urine and evidences of lithæmia. Some authors (Hösslin, Dercum) state that there is an excess of uric acid very uniformly in neurasthenia, and that this uric acid results from a breaking-up of the nuclein of the cells. It has been ingeniously

suggested, therefore, that since the nuclei of nerve cells become smaller and irregular in shape when the cell is exhausted, it is from this source that the uric-acid excess comes. All observation, however, shows that excessive use of nervous tissue leads to an excessive excretion of phosphoric acid rather than of the urates. My experience in studying the urine of neurasthenia is that in the younger cases, with a strong neuropathic taint, it is variable in specific gravity, but, on the whole, rather low; and that the daily amount, as Dr. Herter states, is below the average. It is of a low specific gravity also in neurasthenia occurring after middle life, when the arterial changes of that period begin to set in. In early adult life the urine is more often found to be condensed, as others have observed, and to contain the products of defective metabolism. The urine thus shows either a weakened and slowed-up nitrogenous metabolism or a perverted metabolism. The important things to determine, then, in examining the urine, after excluding such evidences of serious change as albumin and sugar, are: the specific gravity and the daily amount, the amount of phosphates, the amount of urates and uric acid and their relation to each other, and finally, the presence of indican or other products of perverted nutrition and digestion. I do not find indican very often, and practically never in the light urines.

The digestion of neurasthenics is often more or less affected, and a large proportion of them are probably treated mainly for their stomach conditions. I do not, however, usually find cases of serious and genuine gastric disturbance. In the majority, under proper treatment and proper diet, the tongue soon cleans off, and the patient complains relatively little of the stomach, though his nervous symptoms continue. The neurasthenic, it is true, has always a feeble digestion, and has to take great care of what he eats and drinks, but when he is put upon the kind of diet that he should take the stomach gives relatively little trouble. This is especially true of the younger cases. The common form of dyspepsia is one which is associated with acidity, flatulence, some epigastric uneasiness, and constipation. The tongue is often furred, there is a disagreeable taste in the mouth, and frequently anorexia. It is only in patients who have abused themselves with alcohol or tobacco or excessive indulgence in sweets, or with ravenous feeding, that worse conditions are found. In people of more advanced age, however, feebleness of digestion is often associated with a relaxation of the stomach and intestinal walls, and a great deal of atony of the whole intestinal tract. In these cases, which we find particularly often in women, there may be a weakness of the abdominal walls, and with it a cer-

tain amount of prolapse of the large bowel and stomach, with a great many distressing symptoms resulting therefrom. This condition has been described by Glénard under the name of "enteroptosis," and it undoubtedly is an important factor in keeping up the neurasthenia of some women in adult and middle life.

Among the most serious, though fortunately rare, symptoms of neurasthenia involving the digestive tract is the condition known as "mucous enteritis." This trouble generally attacks women rather than men, and usually women between the ages of twenty-five and forty. It comes on after the patient has become exhausted by prolonged domestic cares and fashionable dissipation, or some shock. It is one of the earlier symptoms of the nervous weakness, and begins with abdominal pain, followed by attacks of diarrhœa, in which tubular casts are passed, or portions of such. This diarrhœa is painful, colicky, and alternates with periods of constipation. There is, in my experience and in that of others, a somewhat spastic condition of the bowel, as though it were irritated and closed down upon the contents of the intestine. The term *mucous enteritis* is not strictly a proper one, since microscopical examinations and autopsical reports show that the substances thrown off are not mucous mainly and that there is no actual inflammation. The casts that are found in the stools are composed principally of albuminous substances, the product apparently of the decomposition or disintegration of the epithelial cells of the intestinal walls. While mucous enteritis sometimes occurs in persons who are profoundly asthenic without any decided neurasthenia, yet, in the great majority of cases, it is a symptom of neurasthenia, and can be successfully treated only on such a basis.

The respirations in neurasthenia are generally normal, but shallow and deficient respiratory expansion sometimes exists. In women particularly I have often found that there was an actual inability properly to expand the chest and inflate the lungs.

The temperature is normal, and a very variable temperature of the skin is simply dependent upon vasomotor instability.

The composition of the blood is often quite normal. Hösslin finds that even in those patients who appear to be anæmic there is a normal amount of hæmoglobin; however, anæmia certainly exists in many cases, and there is no question that the use of iron is often of great benefit.

Variations in the weight of the neurotic often occur. Neurotic patients may gain or lose ten or twenty pounds within a year or two. The secretions of the skin are usually increased, and the patient sweats easily and profusely. In other cases of a less irritative

type the skin is inclined to be dry. Its nutritional condition is poor, the hair falls; and according to Beard there is a tendency to early decay of the teeth.

The foregoing description of the general symptomatology of neurasthenia is likely to confuse the reader on account of the multiplicity and wide extent of the symptoms. It is quite true that few neurasthenics have all of the symptoms just described, and it is still more true that in most of them the patients have certain leading and dominant symptoms which annoy and depress them, and that the larger proportion of the manifestations of the disorder are trivial to them, as they are to the physician.

The Different Forms of Neurasthenia.—The peculiar type of neurasthenia depends mainly upon the age, the sex, and the hereditary endowments of the individual.

Primary Neurasthenia.—Neurasthenia appearing at the time of adolescence is much more apt to be associated with a primarily weak nervous constitution. The mental symptoms are mainly dominant, and the malady takes more often the character of a hypochondriasis, with some fixed ideas, or morbid fear, such as suggest an incompletely developed paranoia. Naturally, also, at this time, sexual ideas and sexual symptoms very largely predominate.

Hystero-Neurasthenia.—In women neurasthenia at this period is often associated with hysteria, and the French term *hystero-neurasthenia* is frequently a very apt one for the condition. Nervous and irritable women are usually *hystero*-neurasthenic. In other cases, women suffer from a great deal of pain along the back, and that particular condition, known as "spinal irritation," complicates the neurasthenic state.

Acquired Neurasthenia and Lithæmia.—It is during the mature, active life of men and women that the more typical forms of neurasthenia occur. In these patients the element of heredity is less marked, while the extrinsic causes of neurasthenia, such as excesses in eating and drinking, shocks, injuries, poisons, syphilis, and gouty tendencies, all come much more into play.

Climacteric.—In neurasthenia developing in middle life and at the period of the climacteric, the disease is associated with the natural symptoms that come from beginning degeneration of the arteries and a diminished resistance of the body generally. There is a greater physical weakness, and we often see at this time, also, neurasthenia associated with much vasomotor disturbance, or with the psychoses, such in particular as melancholia.

Traumatic Neurasthenia and Hystero-Neurasthenia.—After receiving an injury which is often but slight, but which is usually

accompanied by a great deal of fright and emotional disturbance, the patient goes to his home feeling perhaps a little nervous and shaken, but not suffering to any great extent. He goes to bed and sleeps; he wakes up the next morning feeling not quite so well as usual, but congratulating himself, perhaps, on having gotten off so easily. He resumes his work and finds that he can do it, though with not quite so much ease as usual and he very likely suffers from some pain due to a strain or bruise that he has received. In a few days—almost always within a week—he begins to notice that he is more nervous than usual, that little things irritate him which did not do so before, that his head seems somewhat confused, and that the effort to work is wearying. His sleep is disturbed, and he wakes up in the morning unrefreshed by his night's repose. He becomes somewhat despondent over his condition, and thoughts of paralysis or some other serious ailment annoy him. His head aches, the pain being more or less constant and diffused, and located usually over the forehead or at the back of the neck. He has unpleasant sensations in the head, such as that of constriction or pressure or scalding feelings. His back also is continually painful, and walking increases it. His nervousness becomes more marked, and close examination shows a little, fine tremor in the hands. He has also sometimes creeping sensations over the body or numb feelings in the extremities. He tires very easily. He is emotional, and becomes more despondent as the days go on. Sometimes he has spots before his eyes, noises in his head, or ringing in the ears. Reading is laborious and increases his headache; so also does attention to work. His appetite becomes capricious and his bowels are constipated. He suffers somewhat from flatulency and dyspepsia. His heart palpitates easily, and the pulse is a little accelerated. Sometimes for a few days there is a little weakness about the bladder or irritability of that viscus. His sexual power is diminished; his circulation seems rather poorer than usual. Very slight excitement produces sweating of the hands or coldness of the extremities. He loses a little flesh.

These symptoms may be several weeks in developing, and during this time he may perhaps consult a lawyer about his case. If so, the anxieties of litigation begin to add to and intensify his troubles. He consults a physician, and the physician finds the subjective symptoms that I have mentioned. Objectively, when examined, the physician will discover that the muscular power is somewhat weakened, that there is a certain amount of the fine tremor perhaps in his hands. The knee jerks and elbow jerks are exaggerated; there are tender points along the spine and upon the

head. In making him stand with his eyes closed there is a certain amount of static ataxia discovered. The pupils are often dilated and mobile, and examination of the visual field shows sometimes a slight contraction, at other times the "shifting type" already described. In many cases a degree of peripheral retinal anæsthesia will be discovered. The pulse will be found accelerated, and pressure on a tender point may send it up very rapidly; a slight exertion will also accelerate it. There will be something apparent in the physiognomy of the case which shows the man to be in a nervous and asthenic condition. Sometimes the pains from which the patient suffers in the back and the weariness in the limbs are so great that he remains a good deal of the time in bed. In all cases he will assert most positively that he is unable to work or to take that interest in his affairs that he has previously done. In a good many cases there will be added to the foregoing picture a number of symptoms due to some local injury: for example, the arm may have been wrenched or bruised, and the result may be a certain amount of neuritis and weakness or pain in that member; in other cases the back may have been so severely sprained that the typical symptoms of spinal irritation ensue, and this is particularly apt to be the case when women are injured; in other cases, again, the legs may have been hurt to such an extent that a sciatica or some other form of neuralgia develops.

The foregoing symptoms, varying in amount and degree, will last, with little change, for a very long period of time. If the case goes into litigation, there is added the worriment occasioned by having to go through the disturbing experiences of trial by jury. In many cases, after the trial has been settled and damages awarded or otherwise, the patient begins to mend, and in a certain proportion of cases he gets completely well. This is not invariably the rule.

Spinal Irritation (Cerebro-Spinal Irritation).—Spinal irritation is a form of neurasthenia in which, associated with the general neurasthenic symptoms, are certain special, painful symptoms, related chiefly to the sensory nerves of the spine. These cases have in the past been described under the head of "spinal anæmia" and "hyperæmia." They may develop in traumatic neurasthenia. The patients are usually young women, between the ages of sixteen and twenty-five. The trouble is sometimes brought on by injuries or by a physical overstrain. Sometimes it seems to be associated with a natural weakness of the spinal muscles and a consequent curvature. Sometimes it follows acute infectious diseases. The patient begins by complaining of pain in the back—usually in the lower part—and also in the back of the neck. These pains occur on

standing or walking, or any exertion, and are so severe that the patients in the course of a few weeks or months give up attempting to walk about. They get relief and comfort in bed, and so they go there and remain. The pains are of a heavy, aching character, increased until they become very sharp when attempts at movement of the trunk are made. There is a great deal of tenderness to pressure along the spinal processes, some of these processes being much more sensitive than others. The most sensitive points are usually in the back of the neck and the upper dorsal vertebræ, and down in the lumbar region. There is some pain also upon pressure alongside of the spinal processes. Painful points often vary, and even in a single examination the patient may complain, and complain honestly, of different sensitive vertebræ. Pressure on these points does not often bring out visceral symptoms, as the brothers Griffin taught. The patients suffer much from headaches. The arms are often weak, so that attempting to sew or write or hold a book causes pain in the neck and shoulders. The legs are also weak and the circulation is poor. There is sometimes palpitation of the heart and precordial distress. A certain amount of dyspepsia is always present, and constipation is the rule. The patients often have attacks of vomiting, and attempts to feed them require much care. The menstrual functions become irregular. The patient grows weaker and often becomes bedridden, especially if little attempt is made to overcome the symptoms by voluntary effort and attention to nutrition. These patients generally get well in from one to three years, but occasionally they sink into permanent invalidism. The symptoms are quite as much due to mental sensitiveness and disordered cerebrum as to any local spinal trouble, and the term cerebro-spinal irritation is a more correct one.

The Anxiety Neurosis, or Neurasthenia with Fixed Idea.—Sometimes neurasthenia is associated with some single idea that becomes fixed in the mind, and worries and harasses the patient through every moment of the waking hours. This idea is always one of a depressing character, and usually one associated with either remorse or fright. For example, one patient of mine had for one or two years an ordinary type of neurasthenia, with a simple nervousness, depression, insomnia, and cerebral paræsthesia. After she had been well for a few years the trouble returned, this time with a fixed idea that during her early life she had committed a very wrong act. The act itself was a trivial one, connected with the taking of a dose of medicine to bring on her courses. But no amount of assurance could entirely relieve her from the distress caused by the continual presence of this remorseful idea.

In other instances a patient will have a neurasthenia following some severe domestic calamity or some shock or injury. Associated with the general neurasthenic symptoms, may be an intense fear that she is going to die from some heart trouble and the patient is continually running to her physician, and feeling of her pulse, under the apprehension that she may drop dead. This condition is not one, strictly speaking, of hyponchondriasis, for the emotional disturbance is much stronger and more dominant than the intellectual one. The patient quite appreciates the unreasonableness of her foreboding, and in her mind believes the assurances of her physician that her heart is perfectly sound; but there is, despite all this, a distress which destroys her peace of mind and makes her nervous, sleepless, and in every way neurasthenic. Such patients do not have other symptoms of a melancholia, although these types of neurasthenia are sometimes associated and appear to be almost abortive forms of melancholia. They, however, do not lose flesh; their appetite may remain good, the tongue is not coated, they have no suicidal ideas, nor do they have the persistent insomnia and the agitation of true melancholia. Neurasthenics with morbid fears of places (agoraphobia, claustrophobia), of dirt (mysophobia), etc., and neurasthenics with the doubting mania, are not true cases of neurasthenia, but the subjects of a psychosis.

Angiopathic Neurasthenia.—There are some cases of neurasthenia in which the vasomotor symptoms are extremely prominent. I do not refer now to Basedow's disease, which represents perhaps in a typical way a vasomotor neurasthenia, but to certain cases in which the innervation of the blood-vessels seems to be especially impaired. I have given histories of several cases of this type, under the head of "angiopathic neurasthenia."

The patient has the general symptoms of neurasthenia, but in addition he has the special symptoms which consist of a sense of pulsation or beating, which involves the whole body. The tension of the pulse is low, the rate normal or slightly accelerated. He does not have palpitations of the heart, as in Basedow's disease, and there is no particular dyspnœa on exertion. The skin usually shows a striking degree of dermography, and there is an epigastric pulsation, as well as pulsation of the carotid.

Neurasthenia Gravis.—In instances which are fortunately very rare neurasthenia assumes a very severe and serious type of exhaustion. The patients suffer from the typical symptoms in much the ordinary way, but the degree of weakness is very much exaggerated. Such patients have not only headaches and disturbed sleep, pains in the back and paræsthesiæ, digestive disturbances, and

mental depression, but they speedily emaciate to a considerable extent. They take food in fair amounts, but it gives them no strength. The most careful applications of the "rest cure" secure for them only temporary benefit. They cannot walk far without intense fatigue and exhaustion, with subsequently severe headaches, or even attacks of vomiting and diarrhœa. Despite closest examination, no distinct signs of organic disease can be discovered, and I have known such patients to go on into a permanent and hopeless invalidism which has lasted for many years. In these cases there is not a hysterical or even large hypochondriacal element. No amount of suggestion or "mind cure" has much effect upon them. They are not, in other words, hysterical, bedridden women, but often men who have reached or passed the middle period of life, and the condition is one suggesting a premature senescence of the nervous tissues.

Pathogeny and Pathology.—Victims of neurasthenia are persons who in all cases have either inherited or acquired a nervous system with lessened power of resistance. In the vast majority of cases I believe that inheritance is the cause of this weak nerve structure. Such inheritance may be very slight, and, if the patient lives with reasonable care, he has good health and lives to an old age. Under the influence of severe and depressing agencies, or of poisons or infections, however, this resisting power of the nerve cells is weakened. The person then is ripe for an attack of nervous exhaustion.

It seems probable that an inherited tuberculous taint in a measure prepares the system for nervous prostration. Among acquired diseases syphilis undoubtedly impairs the physical strength and makes the person predisposed to neurasthenia. So, I believe, does excessive indulgence in alcohol, tea, and tobacco, and I would add an extreme indulgence in the carbohydrates, such as candies, sweets, and pastries, of all kinds, when taken continuously in excess of a normal ratio.

Dr. C. F. Hodge has shown that when the nerve cells are fatigued by persistent work or electrical stimulation, the nucleus of the cells decreases in size, has a jagged, irregular outline, loses its open and reticulated appearance, and takes a darker stain—that the cell protoplasm shrinks slightly in size and stains more feebly. It is a fair inference that human beings who continually and for a long time fatigue their nervous system finally get their cells into a like state and so disorganize them that they are no longer repaired properly; cell bodies and nuclei become permanently shrunken and lose their normal anatomical structure. This view explains certain forms of neurasthenia that come on gradually as the result of per-

sistent overwork or abuse of the nervous system, with bad feeding and stimulation.

A considerable number of cases, however, including most of the traumatic forms of neurasthenia, come on suddenly as the result of a single severe shock. Here we must invoke some other agency, and this I take to be the vascular system. Under the influence of intense and sudden emotions of the depressing kind, the vasomotor centre and the whole vascular mechanism go through a kind of convulsion, and this convulsive disturbance is a thing which the vasomotor system of those predisposed to neurasthenia is unable to withstand. The nerve cells connected with it are so weakened in their nutritive and functional power that the blood is not carried regularly and normally to the nerve centres in the way to which such centres have been accustomed; hence the nerve cells become impaired in nutrition and functionating power.

Another factor undoubtedly exists in the production of neurasthenia, and that is the irritation of the nerve centres by poisons generated within the body. We know that in certain forms of digestive disorder poisons are probably absorbed into the blood, and we know also that in gouty and lithæmic states the uric acid and other products of defective metabolism poison the system and induce many of the symptoms of neurasthenia. There is, therefore, this element of autotoxæmia which enters measurably into the production of neurasthenia. The subject, however, has yet to be worked out into definite shape. When a person has suffered from neurasthenia for a considerable time, there are, no doubt, certain more or less permanent changes in the body; at least we note that catarrhal conditions of the stomach and bowels may become permanent, and that anæmia may be present. In cases occurring in persons advanced in life, arterial changes become more rapidly pronounced than in healthy persons. In fact, a prolonged neurasthenia, with the accompanying worry and mental depression, no doubt hastens and accentuates degenerative vascular changes. Dercum has suggested the name "terminal neurasthenia" for that condition of chronic nerve exhaustion in which anatomical changes have become fixed.

Diagnosis.—Neurasthenia is to be differentiated from the following conditions: hysteria, major and minor; hypochondriasis; melancholia; the beginning stage of general paresis; simulation; the reflex effects of some gross bodily disease.

Hysteria major is distinguished by the presence of the stigmata of that condition and the periodical crises which occur. In the ordinary or minor forms of hysteria the patient does not suffer from

any of the classical symptoms of neurasthenia; she often sleeps well, has no persistent headaches, has a good appetite, and has none of the characteristic paræsthesias and cephalic sensations; she is mentally active and alert, and often gay and cheerful, and is physically strong. The neurasthenic, on the other hand, is generally depressed and serious and greatly concerned in regard to her condition. She is docile and quite willing to do everything possible to get well. She has no severe emotional crises, and none of the globus or the clavus pains. Hysteria minor may be associated with neurasthenia, and in women this is not infrequently the case. The French have for this combination the term "hystero-neurasthenia."

In hypochondriasis the patient suffers from a purely mental malady. There is almost always a history of hereditary taint, and the patient himself usually shows somatic signs of degeneration. He has few of the stigmata of neurasthenia, and is mainly occupied with a fixed idea concerning some special bodily ailment. It is this isolation of mental symptoms, the hereditary taint, and this very marked evidence of the purely psychic disturbance which set off hypochondria, as at present understood, from a neurasthenia. Hypochondriacs, it may be added, are bodily well or at least are able to undertake physical exertions, which neurasthenics cannot do. Here, again, however, it must be borne in mind that a person starting with neurasthenia may finally end up with a form of hypochondriasis; that is to say, he may be practically cured of the asthenic symptoms, but his mind has become disturbed by his painful experience, and he settles down into a mild grade of hypochondriasis.

The early stages of mild forms of melancholia simulate neurasthenia. This is so much the case that some authors have described neurasthenia as an abortive form of melancholia. We have already referred to this under the head of "neurasthenia with fixed ideas," and we there pointed out some of the distinguishing points which enable one to recognize melancholia. The loss of flesh, persistent loss of sleep, rapid pulse, motor restlessness, and extreme mental depression, with delusions and suicidal ideas, are the signs which enable one with very little difficulty to distinguish melancholia. This latter disease, also, is found to simulate neurasthenia mainly in women who are approaching the climacteric.

In the early stages of general paresis the patients suffer from neurasthenic symptoms. They find that they are no longer able to work as they did before, their sleep is disturbed, they are excited, forgetful, and nervous. They have not yet developed many of the physical symptoms perhaps; hence their condition suggests and is

often mistaken for a simple nervous breakdown. The condition is much more perfectly simulated when the patient has been taking a good deal of stimulation, in order to keep himself up to the mark. A careful examination, however, soon reveals the true nature of the trouble. Even in the early stages of paresis some evidence of failure of memory and of the power to write and spell correctly, with expansiveness of ideas, will be found. Besides this, a physical examination will show extreme tremor of the hands, tremor of the face and tongue, and exaggerated reflexes. The pupils also will often be found to be unequal. It is true that facial tremor, tongue tremor, and unequal pupils occur in neurasthenia, but they are rare and not so marked.

A patient may be suffering from a number of bodily ailments, and if this person be at the same time of a somewhat nervous constitution the condition may resemble neurasthenia. Those persons having a very feeble digestion, with dilated stomach and an atonic condition of the alimentary tract, may get depressed, fretful, and sleepless; so a person suffering from some chronic uterine or ovarian or bladder trouble may present many symptoms of nervous irritation. It must depend largely upon the good sense of the physician to measure the importance of the local troubles as compared with those of the general symptoms. I believe that the fully developed type of neurasthenia is rarely brought out by local disease alone. Still, I have seen cases with neurasthenic symptoms cured for a time by washing out the stomach, and enormous relief to the nervous irritation to result from treating the condition of the blood or relieving the uterine disturbances.

Course and Prognosis.—There is such a thing as acute neurasthenia. This follows prolonged debauches and long periods of excessive mental strain, with loss of sleep. Such patients may present all the signs of neurasthenia, and get perfectly well in two or three weeks. Neurasthenia, however, is essentially a chronic disease, and when speaking of it we refer to this type of the disorder. It is a disease which comes on as a rule gradually, developing, however, in the course of a few months. It may, however, come on suddenly after shocks and accidents, and it may develop or follow rapidly after an acute infectious fever. It always reaches its height in a comparatively short time, and runs a course lasting from one or two to seven or eight years. This course is a varying one, and this variation is particularly noticeable when the patient begins to get well. The patient continues to improve for a time and then suddenly falls back, then goes forward again, and thus convalescence progresses. Complete restoration to health is possible and fre-

quent, but the patient always has to take more care of himself than before. As a result of an attack of neurasthenia, men and women who have suffered from it are apt thereafter to lead very saint-like and ascetic lives, and hence they as a rule live long. It used to be said by Dr. Beard that neurasthenics would have a long and happy old age. They pass through the valley of the shadow of death, but the experience may be a profitable, if not a pleasant one.

Treatment.—Naturally, the measure of leading importance in the treatment of neurasthenia is rest, and the problem of how this can be obtained is the first one to confront the physician.

In the severe types of hystero-neurasthenia, especially when it occurs in young women, the application of the "rest cure," which has been so ingeniously elaborated and perfected by Dr. Weir Mitchell, is undoubtedly the best treatment. I do not find, however, that men submit themselves readlly to this measure, and it seems to me to answer best for those neurasthenic women who suffer also from some hysteria and who are reasonably "suggestible" patients. A modified rest cure can often be secured by making the patient stay in bed until after midday lunch or lie down for an hour after each meal, and go to bed early in the evening. Business men will often cut their business hours down one-half if they are allowed still to continue some work. I do not believe, however, that the physician should often use half-way measures, and it is best to impress at once upon the patients the fact that nothing is of so much importance to them as to get well, and to get so that they can take their place at their work again. Change of scene is usually very beneficial to neurasthenics, but travelling is injurious to them. They should be sent to some special place and be made to stay there. A tour along the Mediterranean coast or a trip to Europe often brings them back worse than when they went. Much the same can be said of trips to various places in the South or West. Some of the sanitaria in Germany, some of the places in the Riviera, Egypt and Bermuda, Nantucket, parts of North and South Carolina and Arizona, furnish good resorts for neurasthenics. They generally do better in the mountains, if the altitude is not too high, than they do by the seashore. Dry, windy, sunny climates like those of the Colorado plateau and parts of California and the Northwestern States are too stimulating for most cases.

Much good may be obtained at the numerous sanitaria which exist in this country. Many of these are well conducted and well supplied with all the modern appliances for treatment. It is, however, always a serious thing to send a neurasthenic to a sanitarium,

for the reason that if he stays there too long he becomes contaminated with the atmosphere of invalidism about these places and develops hypochondriacal ideas as to his diet, his liver, his stomach, his sleeplessness, and his various sensory disturbances. In sending a patient to a sanitarium, it is a wise plan to tell him not to stay, under any consideration, longer than six weeks; usually four is better. In the summer time great benefit can be secured by camping out in the woods and living a purely outdoor life, away from all the conventionalities and restraints of civilization.

The diet of neurasthenics, according to the views of most American physicians, should be chiefly a nitrogenous one, and my directions are that the patient can eat meats, fish, eggs, green vegetables, and fruits. Milk can almost always be taken, at least for a short time. There is a certain class of lithæmic patients who do best upon milk, vegetables, and fruit, with practically no meat; these, however, are in the minority. In general, tea and coffee, alcohol and tobacco, are to be entirely prohibited, but this is not an absolute rule. In some cases coffee is beneficial, in some tea does no harm, and in others a small amount of whiskey or dry wine and a cigar are also harmless. The physician has to determine this by the reactions and habits of the patient. Neurasthenics usually drink too little water and it is wise to prescribe a certain amount for them. Four or five glasses of water, which may be either plain or alkalinized, are to be taken daily. I find no special advantage in the various much advertised lithia and spring waters. In dyspeptic patients the meals should be small in amount and taken at frequent intervals; three light regular meals a day and a little food in between form a regimen which usually answers well.

Hydrotherapy, electricity, and massage are all measures which prove of service to the neurasthenic. Of these, hydrotherapy is the most useful, though its value can be overestimated. The ordinary prescriptions consist in the cold sponge bath every morning, and, if it is practicable, the use of a Charcot or a Scottish douche every other day. For women wet packs with massage are sometimes helpful, particularly in cases in which there are a great deal of nervousness and motor irritation. At night a lukewarm bath, at a temperature of 95°, for ten minutes, sometimes relieves the paræsthesia and sleeplessness.

Massage seems to me of not very much use in men, but it is often grateful and helpful to women, and when a great deal of rest is to be enforced it is essential to employ it for both sexes.

Physical exercise is an agent of enormous value in neurasthenia, and the advent of the bicycle has done a vast deal of good in reliev-

ing this condition. Horseback riding is probably just as efficient, but much less practical. Many persons are greatly wedded to the exercise of walking, and it seems best to fit their needs. It is, however, a kind of exercise which does not take the patient's mind off himself, and does not develop the respiratory functions so well as other measures do. Golfing fills in this lack, and this sport will doubtless be of service in neurasthenia.

The drugs of most value are the bromides, nux vomica, mineral acids, quinine, iron, valerian, the coal-tar antineuralgics, the hypnotics, and saline and akaline laxatives, and salicylates.

The bromide of sodium or potassium should be given in small doses; it should be kept up for a limited time and then gradually reduced. At the same time or later the patient may be given a tonic mixture containing such drugs as the symptoms suggest. Quinine must be given carefully, as it causes increase of nervousness in many.

Phosphoric and muriatic acids are the two mineral acids most often of use. These acids are usually better given after meals. The saccharated carbonate of iron or Blaud's pills, if given, should be given generously, *i.e.*, in doses of thirty grains daily. The best preparations of iron are the tartrate of iron and potassium, the carbonate, the citrate, and the tincture. I find no special benefit from the albuminate or the peptonized preparations.

The foregoing covers in a general way the measures to be used in treating neurasthenics. The physician must seek to secure the complete confidence and docility of his patient. He then uses measures which secure some bodily and much mental rest. He gives to him simple and nourishing food in no excess, and prescribes measures which restore the slowed-up or perverted metabolism.

The Sexual Neuroses and Psychoses.

Of the above disorders the neurologist has to deal chiefly with the vicious habits of masturbation (which may, however, be also a manifestation of disease) and the sexual neuroses, spermatorrhœa and impotence.

Masturbation and Spermatorrhœa.—Masturbation is the name given to the vicious habit of artifically exciting the sexual organs. It is very common among boys and less common but present among girls and adult men and women. It is usually only a vice due originally to low associations and teachings among children. In some cases it is a disease or the symptom of a neurotic or insane constitution.

Etiology.—It is most common between the ages of fourteen and eighteen, but may begin earlier. Even infants and very young children sometimes masturbate, usually as the result of some local irritation which leads them to rub the genitals. A tight prepuce, eczema, or worms may lead to the habit, but it is usually taught by a companion. The practice sometimes attacks schools almost like an epidemic, for in every institution a certain per cent of the boys are sexually precocious or vicious, while the others are ignorant and innocent of the evils of the practice. Masturbation is relatively rare after twenty, but is practised by some throughout life even up to old age.

Results.—Masturbation as ordinarily practised leads after a time to a feeling of malaise, mental depression, disinclination to work, study, or to enjoy one's self as before. The appetite is a little impaired, the extremities easily get cold and perspire readily. Peculiar numb feelings are felt in the hands and feet. There are an unnatural nervousness and irritability, and the power of concentrating the mind is a little weakened. The patients often have dilated pupils and hyperæsthetic skin. After a time nocturnal emissions occur. The organs become irritable and slight excitement causes erections. These symptoms may be slightly marked and pass away in a day or two, or until another indulgence occurs.

Masturbation is sometimes done to an extraordinary extent, even daily or twice daily for a considerable time. After a while the young man begins to find that he is not well and realizes that this habit is hurting him. Then if he be sensible and of healthy constitution he stops. Others are frightened out of it by friends or by reading the terrorizing stories printed in quack advertisements and circulars. Sometimes the fright thus caused leads the unhappy youth into a condition of hypochondriasis, which is helped on by the occurrence of nocturnal pollutions and the nervous debility resulting from his past indiscretions. In other cases in which there is a decided neurotic history, a genuine neurasthenia of a sexual type develops and annoys the patient for years.

Masturbation rarely leads to insanity and is oftener a symptom than a cause of such disorder. It is occasionally the cause of epilepsy. When this is the case the convulsive attacks are likely to put on a hysteroid phase and are accompanied by peculiar co-ordinated convulsions and emotional disturbance. Masturbation is the common cause of hystero-epilepsy in women.

Diagnosis.—Many victims of the masturbation habit who have come to recognize its evils and tried to stop it develop a hypochondriacal condition, and feel sure that there is something in their faces

which reveals to the world their trouble. This is not the case. But there is a certain physiognomy which in a measure characterizes the masturbator to such an extent that an experienced observer can detect it. The pale, pasty complexion, moist, furtive eye, dilated pupil, listless, restless, and depressed manner, the wet, flabby palms, and hyperæsthetic skin, all help to tell the story. Locally the penis is often reddened and more or less turgid, the scrotum relaxed, and a varicocele may be present. Examination of the urine may reveal spermatozoa. The urine also is almost always of rather low specific gravity, and contains a great excess of phosphates, both earthy and alkaline.

Treatment.—The patient must be told plainly the necessity of stopping the practice. He must be impressed, but not terrorized. He should be kept out of doors at vigorous physical exercise, for sedentary and solitary work is always bad for such cases. He should be made to take cold-water baths and should sleep on a hard bed with light covering. He had better sleep with some one whose presence may exercise a controlling influence. He should not eat heartily at night, never just before going to bed, and what is still more important, he should not drink before going to bed. Sometimes it is well to have him wakened at an early hour in the morning, when he should empty his bladder; for emissions occur often early in the morning and are promoted by the irritation of a full bladder.

Locally cold-steel sounds may be introduced and allowed to remain for ten minutes three or more times a week, or the psychophor or Ultzmann's short catheter may be used. In bad cases with a great deal of prostatic irritation, local applications of nitrate of silver are needed. Internally a mixture of tinct. opii, tinct. camph., and tinct. lupulin may be given at night, the ingredients being somewhat varied in amount to suit the case. Bromides, chloral, atropine, and salix nigra are also drugs which are often useful. The mechanical measures which have been devised for preventing erections, such as rings with sharp teeth, are rarely needed and rarely useful. They may even do harm by directing the mind to the affected function.

I do not believe it right for the physician to prescribe fornication. It is not safe nor curative, apart from the moral aspect of the matter. It has always struck me also as pretty small business for a man purposely to select a wife to relieve him of the results of a weak will and vicious sensual indulgence. If marriage comes in the natural course of events, as it often does, so much the better. But to select a wife as a remedial agent for masturbation is unjust to the woman and a confession of moral and mental feebleness.

Man is distinguished from the brute by his self-control. Let him bear this fact in mind and raise himself above the animals by a determined effort of the will. Pure thoughts and chaste associations, vigorous physical exercise, and a resolute effort to act a manly part will always be successful.

Traumatic Nervous Affections (Traumatic Neuroses and Psychoses, Spinal Concussion).

The present tendency of neurology is to deny the existence of any special nervous affection produced by trauma or shock. There may follow from these causes:

1. Surgical injuries.
2. Neurasthenic states.
3. Hysterical states.
4. Hemorrhagic, inflammatory, and degenerative diseases.
5. Combinations of the foregoing.

These troubles may follow not only railway but other injuries, but are especially liable to follow those associated with intense fright.

2. Traumatic neurasthenia, or "traumatic neurosis," "railway spine," does not differ from forms of neurasthenia produced by other causes, except that with it there may be certain sprains and surgical troubles. Its special symptoms are described under the head of neurasthenia.

3. Traumatic hysteria is a rare affection in this country. It does not differ from hysteria produced by other causes, except for its sudden onset and occasional surgical complications. It is usually a hysteria major and has the characteristic stigmata of that type. In this city electrical injuries and frights have produced some classical cases of hysteria major.

4. There is considerable evidence that in some rare cases traumatism may produce minute multiple hemorrhages throughout the nervous centres. In such cases there are usually neurasthenic or hysterical symptoms and in addition symptoms of organic disease.

In the majority of cases the symptom complex is something like this (Knapp): "The patient has headache and vertigo; he is depressed, iritable, and hypochondriacal, with a diminished power of application; he may have some visual disturbance; he often has a contracted field of vision and occasionally optic atrophy; there is some tremor and perhaps inco-ordination; he has anæsthesia, usually not limited to one-half of the body, and with it numbness and prickling; his movements are slow and weak; his tendon reflexes

are exaggerated; there is often some lack of control over his bladder; and he may have pain and stiffness in the back from muscular strain." The symptoms eventually resemble a disseminated sclerosis.

Massive hemorrhages and serious mechanical injury of the nervous centres may be also produced by injury.

Finally, it is a well-known fact that traumatisms may excite in the predisposed locomotor ataxia, inebriety, insanity, or may lead to the development of a cerebral tumor.

It is the mental impression, the shock, much more than the physical injury, which produces the functional neurosis or psychosis.

The symptoms may appear soon after the accident, or after a period of relative health lasting some weeks the neurosis gradually develops.

The most important practical point in connection with the subject is the diagnosis and the elimination of malingering. This is additionally difficult for the reason that the hopes and anxieties depending upon litigation tend to cause introspection, exaggeration of symptoms, and unconscious bias even in the most honest. The opinion among American neurologists tends to favor the seriousness of traumatic neuroses. While malingering is not rare, yet if the patient has really a traumatic neurasthenia or hysteria the disease may not be a trifling one. Careful research, however, often tends to elicit the fact that previous to the injury the patient was an alcoholic, syphilitic, or neurotic, and perhaps had already the beginnings of his alleged traumatic disorder. In no part of clinical medicine is a careful and searching examination and weighing of symptoms more urgently called for. The methods of carrying out such examinations are given elsewhere. Special methods for testing anæsthesia are sometimes needed. The two sides of the body should be tested simultaneously with concealed needles, beginning on the trunk, or the faradic current with a double-pointed electrode may be used. There are few patients who can successfully deceive in an examination covering all the special senses.

The treatment of the neuroses calls for no special notice.

Exophthalmic Goitre (Basedow's Disease, Graves' Disease).

Exophthalmic goitre is a chronic glandular neurosis characterized by rapid heart beat, enlargement of the thyroid gland, protrusion of the eyeballs, and various neurasthenic and vasomotor symptoms.

Etiology.—The disease occurs much oftener in women than men

(four to one). It is a disease of early adult life, occurring chiefly between fifteen and thirty-five, very rarely in childhood, and never after fifty.* It is apparently more common in the Anglo-Saxon race, but is not very frequent in America, at least in the Eastern States. I am informed that it is rather common in the Northern Central States. There is very rarely any direct inheritance of the disease, but the family is often a neuropathic one. As a rule, the patient is of a neurotic temperament. Anæmia and debilitating diseases promote its development. Goitre and heart disease do not seem to predispose to it. The most frequent exciting causes are powerful depressing emotions and severe physical exertion. Rarer causes are injuries and infectious diseases, such as measles, scarlet fever, and pneumonia.

Symptoms.—The disease usually begins gradually and the first symptom is in most cases rapid heart beat and palpitations, accompanied with some nervousness and tremor. The next symptom is enlargement of the thyroid gland, and at about the same time the eyeballs begin to protrude. This order of development does not always take place, and occasionally one of the three principal symptoms is not present. The disease is usually one or two years in developing, the heart symptoms being those which continue by themselves longest. With the symptoms mentioned there occur many minor troubles which are more or less characteristic. The patient is usually very nervous and irritable; a distressing insomnia may be present. There is almost uniformly a fine tremor (eight to nine per second) of the hands, less marked in the lower limbs and not present in the face or tongue. The reflexes are exaggerrated. There is a tendency at times in walking for the knees suddenly to give way. The patient rarely has neuralgias, but does have burning or feverish sensations and headaches. The skin is rather reddened and the patient sweats profusely. Pigmentation and vitiligo are sometimes seen, and urticaria may develop. The electrical resistance of the body is much diminished, being 800 to 1,500 ohms instead of 2,000 to 3,000. There is sometimes a dermographic skin, as in other neurasthenic states. The respiratory function is weakened and chest expansion often falls below one inch (Fiske-Bryson). Attacks of a persistent watery diarrhœa occur. Anæmia is usually present. There is occasionally polyuria, more rarely glycosuria. The menses are irregular and amenorrhœa often exists. A slight rise in temperature may occur.

* Among 33 cases at the New York Post-Graduate Clinic (Fiske-Bryson) there were 8 males, 25 females. Ages: thirteen to twenty, 8; twenty-one to thirty, 8; thirty-one to forty, 6; forty-one to fifty, 5; fifty-one to sixty, 1.

The *major symptoms* of the disease are:

Tachycardia and pulsating arteries.
Goitre.
Exophthalmus.
Tremor.

The *minor symptoms* are:

Nervousness.
Sweating.
Insomnia.
Lessened electrical resistance.
Subjective sensations of heat.
Diarrhœa.
Polyuria.

Symptoms in Detail.—Tachycardia is the most constant single symptom. The pulse beats from 100 to 120 per minute usually, but may rise to 160 or even 200. Its rhythm is usually steady; but palpitations occur easily, even without exciting cause. The patient may wake up at night with distressing attacks, something like those of angina pectoris, but the intense pain and sense of impending death are usually absent. The heart is dilated and a systolic murmur is often heard at the base propagated along the arteries. Real organic disease, however, is rare. The arteries are dilated and soft. They pulsate strongly, particularly the carotids. A thrill is sometimes felt over the heart and always over the goitre. The arterial tension is normal or low.

The thyroid gland is usually enlarged symmetrically; later in the disease the isthmus is affected and the three lobes of the gland stand out prominently (Fig. 227). If only part of the gland is involved it is oftenest the right lobe. A thrill is felt over it and a systolic murmur can be heard.

The bulging of the eyes or exophthalmus is usually bilateral and even. If one eye is alone or more affected it is the right. The exophthalmus varies much in degree. It is not usually very great, but may be so excessive as to prevent closing of the lids and to expose the insertions of the recti. The eyeball may be slightly enlarged (one-tenth). The pupils are normal and vision is not impaired, though myopia occasionally occurs. The fundus and visual field are normal. Paralysis of some of the eye muscles is a rare complication. Weakness of the internal recti and exophoria are frequent. The lids show certain peculiarities. One of these, known as *Von Graefe's symptom*, consists in the inability of the lid to follow the downward movement of the eyeball. When the patient is told to follow the movement of the finger vertically downward

the eyeball moves steadily, but the lid catches, as it were, and refuses to follow or does so in a jerky manner. Another symptom, known as *Stellwag's symptom*, is a considerable retraction of the lids, especially the upper one. Both this and Von Graefe's symptom are due to a common tendency of the lids to retract—due perhaps to overaction of the muscle of Müller. A tremor of the lids sometimes occurs.

Course.—The disease progresses slowly. After a year or two it often becomes stationary for a long time. Cases of gradual spontaneous recovery occur. The natural duration of most recoverable cases is two or four years. In those which do not recover the dis-

FIG. 227.—EXOPHTHALMIC GOITRE WITH AND WITHOUT EXOPHTHALMUS.

ease lasts five, ten, or more years. Eventually the patient emaciates, the heart becomes weaker, albuminuria and dropsy appear, diarrhœa sets in, and the patient dies of exhaustion or is carried off by phthisis or some intercurrent disease. Other cases, having improved up to a certain point, remain in this state for years.

Complications.—Mental derangement occasionally occurs in the later stages of the disease. Hysterical crises, epileptic attacks, choreic movements, paralysis of the ocular muscles, muscular atrophy, paralysis agitans, Addison's disease, diabetes, locomotor ataxia, and local œdema have all been observed. With the exception of hysterical attacks these complications are rare.

Abortive Forms.—This name is applied to cases in which only a part of the distinctive symptoms develop. Tachycardia always exists; with it are tremor and moist skin, lessened electrical resist-

ance and nervousness. Or tachycardia and goitre may alone be present.

Pathological Anatomy.—Post-mortem examination of this gland shows that it undergoes a true hypertrophy with increase of vascularity and of the glandular structure. The rational inference is that in life there is an increase in the secretions from this structure. After the hypertrophy has reached a certain stage, the glandular epithelium degenerates and breaks down, forming large acini filled with the colloid secretion. In the nerve centres the changes which have been found are small hemorrhages in the medulla and degenerated nerve cells. In one case of about a year's standing, I found a very marked pigmentation and vacuolization of the cells of the vagus and glosso-pharyngeal nuclei. In another case of six months' standing, no marked changes could be seen in these areas, but there was a spot of softening at the junction of the pons and the cerebral peduncle. This was ante mortem and had led to crossed paralysis just a few days before death. In other cases congestion and small hemorrhages in the medulla have been found. The heart is dilated and enlarged; endocarditis is sometimes present, oftener not; the arteries are dilated.

Pathology.—Some writers now consider this disease primarily one due to a disordered function of the thyroid gland. From my own observation I am led to the conclusion that Basedow's disease is primarily one of nervous origin, but that the thyroid disease leads to excessive secretion, causing the principal symptoms. The nervous tissues require for their proper nourishment and natural functioning a certain supply of the secretion of the thyroid gland. If this is excessive, there is a state of nervousness and erethism, such as we see in Basedow's disease, and if it is diminished there develops a hebetude and depression of nerve function, such as we find in myxœdema. Under the influence of shock and powerful emotion or prolonged strain, there is a certain powerful demand upon the product of the thyroid gland by the nerve centres. The thyroid juice is thrown out in great amount, and in persons of unstable organism a morbid impetus is given to the activity of the gland. It continues to grow and throw out its juice; the overexerted nervous system makes continually more demand upon it, thus acting in a vicious circle. So, while the symptoms of the disease, the nervousness, the insomnia, and the vasomotor disturbances are due to the hypersecretion of thyroid, the primary disturbance is one in the nerve centres. If these can be kept quiet long enough, the demand on their part for this excess of thyroid juice gradually ceases and the patient gets well. This is the rationale of the pro-

longed rest and the use of the bromides and tonics, which are the only things which do much good in the treatment of the disease.

As to the special cause of the different symptoms, it may be assumed that the original enlargement of the thyroid is a vasomotor paresis of its vessels. It is a kind of erection of the organ, due to the sudden demand put upon it by emotional strain and exhausting work. The exophthalmus is also due chiefly to paralysis of the orbital vessels. A tonic spasm of the muscle of Müller is thought to help in producing this symptom. This muscle consists of unstriated fibres originating in the membranous lining of the orbit and inserted into the lids. It is rudimentary in man, and its influence in causing protrusion of the globe must be very small. The deposit of retrobulbar fat is a secondary phenomena. The rapid heart beat is probably due to impairment of the inhibitory fibres of the spinal accessory.

Prognosis.—About one-fifth of the cases get well or practically well. Probably over half the cases, if they can be properly treated, reach a fairly comfortable condition of improvement. The cases in which symptoms come on quickly have the most favorable prognosis. In those with marked exophthalmus and goitre the prognosis is not so good. The duration of the disease in recovering cases is from two to eight years.

Diagnosis.—The disease can be distinguished by the persistent tachycardia, with goitre or exophthalmus, and in its early stage by the tachycardia with tremor, moist skin, sensations of heat, nervousness, insomnia, lessened respiratory expansion, and electrical resistance. A symptomatic Graves' disease may sometimes be caused by a goitre pressing on the vagus or sympathetic and causing irregular heart beat and perhaps exophthalmus. In these cases the history of a long-standing goitre exists, the heart's action is irregular, the exophthalmus is usually partial and one-sided. In abortive forms it is necessary to have tachycardia and at least one other of the four major symptoms to make a diagnosis.

Treatment.—Rest is the most important single thing. The patient should be put to bed or kept on the back for one or more months. Freedom from excitement and worry must be enjoined. No especial diet is needed, nor do climatic influences or baths or mineral waters have much effect. Some cases are said to be improved, however, by removal to heights of one to three thousand feet. In most cases a sea voyage is the better change if one is made.

The drugs used are numerous. The most efficient are tincture of strophanthus in doses of fifteen to forty drops daily; iodide of

potassium or the syrup of hydriodic acid; arsenic and bromide of potassium used together; quinine, mineral acids, and iron, used together. Dilute phosphoric acid is often very useful. Other remedies are tincture of aconite in ♏ v. or aconitia in gr. $\frac{1}{200}$ doses, tincture of belladonna increased to the limit of tolerance, tincture of cactus grandiflorus in ♏ x. to xx. and tincture of veratrum viride, ♏ x. to xxx. dose, and the picrate of ammonium, gr. i. to ij. t.i.d. Of these drugs, strophanthus, aconite, the iodides, bromides, and iron have served me best. Digitalis is of doubtful value.*

Electricity possesses some utility. It should be given, if possible, two or three times daily in the form of galvanism and in doses of two to six milliamperes for ten minutes. The technique is as follows: positive pole on back of neck, negative drawn along course of vagi in the neck; each side two minutes. Same with positive pole placed subaurally one minute; negative pole over thyroid two minutes, negative over cardiac region one minute, positive pole over eyes, negative over thyroid one minute, two milliamperes. The faradic current may be used for general tonic effects or combined locally with the galvanic. The patient should lie down during treatment and remain quiet for an hour later.

For the palpitations, sulphate of sparteine or strophanthus with Hoffmann's anodyne may be used. The ice bag placed over the heart and neck is helpful and may be used systematically. Tonic hydrotherapy is often useful, but should be carefully employed. Surgical treatment until late years has been unsuccessful. Recently many cases have been reported in which cure has been produced by partial removal of the thyroid gland. This measure is yet in an experimental stage. Treatment of the nose has been said to cause disappearance of symptoms, but its utility is very doubtful. Respiratory exercises by which the patient is taught to increase his chest expansion do some good. Mild compression of the lids at night seems to help the exophthalmus, and slight and steady compression of the thyroid gland sometimes reduces its size a little.

* Some writers now lay great stress on the direct treatment of the thyroid gland by rubbing upon it daily the ointment of the red iodide of mercury; or by painting it with iodine.

CHAPTER XXVI.

PROFESSIONAL NEUROSES, OCCUPATION NEUROSES (WRITERS' CRAMP AND ALLIED AFFECTIONS).

Writers' cramp is a chronic functional neurosis characterized by spasmodic, tremulous, inco-ordinate or paralytic disturbance when the act of writing is attempted, and associated with feelings of fatigue and pain.

Etiology.—It is a disease of the present century, and has been particularly noted since the introduction of steel pens about the year 1820. A neuropathic constitution is often present, and sometimes there is a hereditary history. Men are much more subject to the disease than women. The most susceptible age is between twenty-five and forty. It rarely occurs after fifty or before twenty. Clerks and professional writers are naturally much more subject to the disease. Excessive worry, intemperance, and all debilitating influences predispose to it. The chief exciting cause is excessive writing. But this is not all. The writing that is done under strain or a desire to finish a set task is the harmful thing. The style of writing is also an important factor. Writing done in a cramped posture with movements of the finger alone or with the little finger or wrist resting on the table is most injurious. Free-hand writing done from the shoulder according to the American system is least harmful. Shaded or heavy writing with sharp steel pens is also productive of harm. Copying is much more harmful than composing. Authors seldom have writers' cramp. Albuminuria, lead poisoning, exposure to wet and cold, and local injuries are sometimes exciting causes.

Symptoms.—Writers' cramp very rarely attacks a person suddenly. The patient first notices a certain amount of stiffness occurring at times in the fingers, or the pen is carried with some uncertainty and jerky movements are made. He feels a sensation of fatigue in the hand and arm, and this may amount to an actual tired pain. The first symptoms may last for months or even years.

The hand is rested as much as possible; new pens or penholders and new modes of holding it are tried. Often the patient, fearing the onset of the cramp, and as its result loss of employment, becomes anxious, worried, and mentally depressed. Sometimes the trouble is worse when beginning a daily task, and it gradually wears off in a few hours. At other times exactly the reverse is the case. When the disease has reached its highest stage, writing becomes almost or entirely impossible. The moment the pen is taken in the hand and an attempt at using it made, spasmodic contractions of some of the fingers, or even of the arm, occur, the pen flies in any direction, and it is impossible to control or co-ordinate the movements. The rule is that although writing cannot be done, all other complex movements are performed as well as ever. Thus the sufferer from writers' cramp may be able to play the piano, or paint, or thread a needle, or use the hand in any complex movements. This limitation, however, is not always present. Telegraphers, who use to some extent the same muscles as in writing, and who also often have to do a great deal of writing, are liable to suffer from both writers' and telegraphers' cramp at the same time. No evidences of actual paralysis are present in the affected muscles, and there is rarely anæsthesia, but the arm aches and is sometimes tender. Sensations of numbness and prickling are present: in rare cases vasomotor disturbances are observed; associated muscular movements of the other arm or of the neck or face sometimes occur. The hand may tremble on attempting to write or fall almost paralyzed when the pen is taken.

The various symptoms occur with different degrees of prominence, so that the disease has been classed under the heads of (1) the spastic, (2) the neuralgic or sensory, (3) the tremulous, and (4) the paralytic forms. These forms are, however, often more or less mixed.

1. The spastic form is undoubtedly the most common, and it has given to the disease its name. Cramp of some muscle or muscles is present in over half of the cases. The muscles of the thumb and first three fingers are oftenest affected, and in some cases the flexors, in some the extensors, are chiefly involved. In telegraphers' cramp it is the extensors, but in writers' cramp the flexors, that are mainly attacked. The thumb or forefinger or the little finger alone may suffer from the spasms. The pronators and supinators are quite often involved. The spasm is usually a tonic one. With the spasm there is also inco-ordination so far as writing movements are concerned, and this fact is quite as important in producing the bad writing as the spasm.

2. The neuralgic form resembles the spastic plus sensations of fatigue and pain, which are quite severe and are brought on by writing. There may be tenderness along the arm also.

3. The tremulous type, though rare, is very characteristic when present. The patient when attempting to write develops a tremulous movement of his hand and arm. This ceases when his attempts to write cease. The tremor usually affects most the fingers used in pen prehension, but it also spreads to the forearm and may even involve the entire extremity. An oscillatory or lateral tremor, due to involvement of the pronators and supinators, has been observed. The tremor is of the character known as "intention tremor," such as is observed in disseminated sclerosis. It is shorter in range and more rapid than the tremor of that disease.

4. The paralytic form, or that type in which muscular feebleness is the dominant symptom, is said to be rare by Gowers, and this accords with my experience. German writers speak of it as common. In the typical paralytic form the patient, as soon as he begins to write, feels an overpowering sense of weakness and fatigue in the fingers and arm. The fingers themselves loosen their grip and the pen may drop from the hand. Powerful impulses of the will and change in the mode of holding the pen enable the sufferer to continue, but the arm aches and finally is absolutely painful, and weakness and fatigue compel the writer to desist. Sometimes the paretic condition is succeeded by the spastic. Many of the cases of paralytic writers' cramp are not true examples of the neurosis, but are rather cases of neuritis of a rheumatic or other type.

General Symptoms.—Writers' cramp is essentially a motor neurosis, and its leading symptom is the impairment of a motor function. Other symptoms, however, both general and local, are always associated with it. These are mainly (1) psychical and (2) sensory, more rarely (3) vasomotor and (4) trophic.

1. Psychical symptoms. The patient is often nervous, emotional, and mentally depressed at times. He suffers from insomnia and vertigo. Patients are generally unwilling to admit that there is any other trouble than the local one, and only careful examination may bring evidence of constitutional trouble. There are cases of purely mental writers' cramp.

2. Sensory troubles. These consist of pains, sense of fatigue, feelings of numbness, prickling, pressure, weight, tension, constriction, etc. Hyperæsthesia, and more rarely anæsthesia, are also observed. The most common sensory symptom is that of aching and fatigue, and this is usually confined to the arm, and oftenest runs along the course of the radial and median nerves. The cervical ver-

tebræ may be tender, and sometimes patients have a headache in the parietal region of the side opposite the affected arm.

3. Vasomotor, trophic, and secretory disturbances. The condition known as *digiti mortui* has been observed, coming on paroxysmally. It is a symptom which the general neurasthenic state helps to produce. When the nerves are involved decided vascular changes may occur, such as passive congestion of the hand and arm, with swelling and turgescence of the fingers, and a sensation of throbbing. In bad cases the fingers will look as if they had chilblains. Local sweating, dryness of the skin, and cracking of the nails, all are conditions which may follow impairment of writing power from neuritic causes.

Electrical Reactions.—The results of observations upon the electrical reactions of the affected parts are somewhat contradictory. Ordinary tests will, as a rule, reveal very little change. Sometimes there is a quantitative increase, sometimes a decrease, of irritability to both forms of current. The increase occurs in the earlier stages, the decrease in the later. An increase or modification of electro-muscular sensibility has been noted. The electrical examinations, therefore, are only of value in excluding a neuritis or possibly in determining the stage of the disease.

Pathology.—Neuritis is undoubtedly present in some forms of writers' cramp, so called. It is not present, however, so far as external tests go, in the typical neurosis. Nor are there any post-mortem observations throwing light on the anatomy of the disease. We must believe, therefore, that it is a neurosis having no appreciable anatomical basis.

The act of writing is a very complicated one, calling into play numerous sets of delicately innervated muscles. These muscles are employed: 1, in pen prehension; 2, in pen movement; 3, in holding the arm and wrist tense.

1. The muscles employed in pen prehension are the two outer lumbricales, two outer interossei, the adductor muscles of the thumb, the flexor longus pollicis; to some extent the deep and superficial, short and long flexors, and the extensors of the thumb. These are supplied mostly by the ulnar (interossei, adductor pollicis, inner heads of deep flexor of fingers, and inner head of short flexor of thumb). The rest of the muscles are supplied by the median.

2. In moving the pen, if the writing is done mainly by finger and not by arm movements, the muscles brought into play are the flexor longus pollicis, extensor secundi internodii pollicis, flexor profundus digitorum, extensor communis digitorum, and to some extent the interossei. The musculo-spiral and ulnar nerves innervate

these groups about equally. In moving the pen by the "American" or free-hand method there is a very slight play of the above muscles, while most of the pen movement is done by the muscles of the upper arm and shoulder, viz., the teres major, pectorales, latissimus dorsi, biceps, and triceps.

The spinal centres for these muscles are distributed along the fifth, sixth, and seventh cervical segments of the cord. The cells are larger and situated more superficially in the anterior gray horns.

3. Besides these movements involved in pen prehension and in the letter making, a certain amount of muscular tension is exercised in "poising" the forearm and hand and steadying the wrist. The biceps and triceps, the supinators and the flexors and extensors of the hand are here brought into play.

From the foregoing it will be seen that the muscles of pen prehension are most used in all but the free-hand style of writing, since the same groups have a double duty, that of clasping and of moving the instrument.

While writers' cramp is often complicated with some neurotic disturbance leading to symptoms in the affected arm of pain, paralysis, tenderness over nerves, vasomotor disturbances, etc., there can be no doubt that the lesion in typical cases is central, and involves the psycho-reflex centres and indirect motor and sensory paths. Little more can be said of the pathology than that it is an "exhaustion neurosis." The same is true of all the other forms of occupation neuroses, and nothing need be said upon this point regarding them when they come to be considered.

The *diagnosis* of well-marked cases of writers' cramp presents no difficulty. In the earlier stages, however, it may be confounded with a large number of disorders, viz., post-hemiplegic chorea, hemiataxia, progressive muscular atrophy, progressive locomotor ataxia, various forms of tremor, lead paralysis, rheumatoid arthritis, neuritis, cerebral and nerve tumors, and tenosynovitis.

In many of these cases it is only necessary to bear in mind the history of the disease in order at once to reach a safe conclusion as to its nature.

If there is a great deal of pain in the arm, with tenderness along the course of the nerves; if there is decided change in the electrical reactions; if there are sensations of tingling, numbness, etc.; and if the patient shows an absolute loss of power in the various groups of muscles, with some incapacity for doing other acts besides the one with which he is specially concerned, then the trouble is undoubtedly peripheral and due largely to an underlying neuritis. The prognosis in these cases is much more favorable. If, on the

other hand, the disorder comes on in persons who have done an excessive amount of writing; if it is associated with nerve strain; if the electrical reactions are but slightly changed, the sensory symptoms slight, and the motor inco-ordination is marked, limited to the special class of work, and not accompanied with absolute paresis, the disorder is central and needs both a different treatment and prognosis. It is these cases that form writers' cramp proper, although no doubt neuritic and central forms are associated, or the former may run into the latter.

Course and Duration.—Writers' cramp is a chronic disease. It begins insidiously and attacks one group of muscles after another as each is brought into play by new methods of writing. If the left hand is used, that, too, is liable to become affected. The course varies, however; for a time progress may be arrested or improvement set in. When the disease becomes well established it will most often last a lifetime.

Prognosis.—The prognosis is unfavorable, yet not so much so as was once thought. Undoubted cases of complete recovery have been reported, even under unfavorable conditions. The prognosis is much more favorable if the patient begins treatment early and before marked spastic symptoms are present. It is more favorable in the neuralgic forms. Some patients who suffer from a mild form of the trouble manage, by the help of instruments or special pens, to do their work for years. The more acute the disease and the more evidently peripheral and neuritic its origin, the better the prognosis. In over one-fourth of the cases, patients who use their sound arm will not be affected in it.

The facts stated regarding the cause, physiology, and general symptomatology of writers' cramp apply to the other forms of occupation neuroses. A few special details, however, will be given regarding these. The most common and important are musicians' cramp and telegraphers' cramp.

Musicians' Cramp.—Under this head we include pianists' cramp, violinists' cramp, flutists' cramp, and the cramp of clarionet players.

Pianists' cramp occurs usually in young women who are studying to become professionals or who are especially hard working and ambitious. The absurd "Stuttgart method" of teaching the piano, in which the motions are confined as much as possible to the fingers, predisposes especially to this disease. The symptoms are those of fatigue, pain, and weakness. The pains are of an aching character. They are felt in the forearm especially, but extend up the arm and between the shoulders. Spasmodic symptoms are rare. The right hand is oftener affected, but both hands eventually become involved.

Violinists' cramp may attack the right hand which holds the bow or the left hand which fingers the strings, but more often the left hand is affected.

Clarionet players sometimes suffer from cramp of the tongue and of the laryngeal muscles.

Flute players suffer not very infrequently from slight laryngeal spasms. A similar trouble affects elocutionists. The term mogophonia is applied to this type.

Telegraphers' cramp affects especially those operators using the Morse system, which is still the one most widely in vogue. Contrary to the opinions of previous writers, Dr. Lewis believes that this neurosis is not a rare one and is destined to become more frequent. In this city the cramp is not rare, the proportion being about one in every two hundred. The technical name among operators for the cramp is "loss of the grip." In telegraphing, the extensors of the wrist and fingers are called most into play, and hence are most and earliest affected. The symptoms come on very slowly, the thumb and index finger being first affected. The victim finds that he cannot depress the key on account of spasm in these muscles, and he finds most difficulty in making the dot characters, such as h (. . . .), or p (. . . . .), or z (. . . . . .). When the flexors are most affected the key is depressed with undue force and a dash is made instead of a dot. Sufferers from the "loss of grip" generally have writers' cramp also. While spasm is usually present, the disease may show itself simply in pain, paresis, and incapacity to co-ordinate the muscles.

In *sewing-spasm*, which affects tailors, seamstresses, and shoemakers, clonic and tonic spasms attack the muscles of the hands on attempting to use them in the regular work. Tailors who sit cross-legged sometimes suffer from a peculiar spasm on assuming this position. It is possible, however, that these are cases of tetany, and not the functional neurosis under consideration.

Smiths' spasm, or "*hephæstic hemiplegia*" appears to have been observed only by Duchenne and Dr. Frank Smith. It occurs in persons engaged in pen-blade manufacturing, saw straightening, razor-blade striking, scissors making, file forging, etc. In doing this work they have to use a light or heavy hammer, with which strokes are delivered very rapidly and carefully. After a time spasmodic movements occur in the arm used, and the arm falls powerless. As in the cases reported, there are generally hemiplegic symptoms, and also neuralgias, vertigo, and other cerebral troubles, the disease cannot be a pure "occupation" neurosis.

Drivers' spasm has been observed in veterinary surgeons by Dr. Samuel Wilkes.

Milkers' spasm is an extremely rare affection, which was first described by Basedow and seems to occur in milkmaids, never in milkmen.

Cigarmakers' cramp is very rare.

Watchmakers' cramp and *photographers' cramp* are also to be regarded merely as pathological curiosities.

Ballet-Dancers' Cramp.—Under this name certain painful and paralytic troubles occurring in ballet dancers, especially premières danseuses, have been described by Schultz, Onimus, and Krausold. It does not appear that the trouble is really a co-ordinative functional one, but is rather neuralgic or the result of local strain upon the parts.

The list of professional neuroses is made to include, besides those above given, cramps and co-ordinative troubles affecting artificial-flower makers, billiard players, dentists, hide dressers, electrical-instrument makers, stampers, turners, sewing-machine girls, money counters, weavers, painters, and pedestrians.

Prophylaxis and Treatment.—The introduction of typewriters, gold pens, and improved penholders has prevented somewhat the increase of writers' cramp. Stenographers rarely have it unless they write in long hand. Persons who have to write a great deal should use large cork or rubber penholders and gold or quill pens with smooth paper. The best style of writing is that done from the shoulder, but this is a method that bookkeepers and those who have to keep accounts cannot easily adopt. The vertical system of writing which is now being widely taught is to be preferred. Many nervous persons have a bad habit of gripping the pen very tightly and pressing down on the paper with excessive force. Fatigue soon results and painful sensations develop in the arm. Proper attention should be paid to the position of the paper written upon, the height of the desk, the light, and the sleeves of the coat or dress. The paper should be laid at an oblique angle to the edge of the desk, and not at a right angle as many writing-teachers are accustomed to direct. As some cases of "cramp" are undoubtedly cerebral, it is very unwise to attempt any extraordinary exploits in writing or to work with the ambition to put the writing-capacity to the utmost test. Cramp is often dated from days when such extra work is done.

When the cramp is fully developed, the most essential thing is rest, and it is generally best to advise the patient to change his occupation at once. Some rest, however, may be secured by getting a new form of penholder, holding the pen in a different way, using the unaffected arm, or using some form of mechanical appliance. The mechanical appliances are splints, rubber bands around the wrist, and various instruments contrived to prevent spasm and throw the work of writing on new and larger groups of muscles.

Instruments for writers' cramp are very numerous. Those that are of some value are Mathieu's, Nussbaum's, and some modifica-

tion of Cazenave's (see Figs. 228, 229). All the various instruments have been of service, or have even been curative in some special cases, but not too much must be expected of them. As a rule they are only palliative. A cheap instrument that may prove satisfactory is that of Mathieu.

In the medical treatment of writers' cramp, two important agents are massage and electricity.

By massage only very mediocre results were obtained until greater attention was drawn to it by Mr. J. Wolff, a writing-master of Frankfort-on-the-Main. This gentleman has cured many cases, though not all that he has treated (Berger), and he has secured many testimonials for his method. The treatment, as described by Schott (G. W. Jacoby), consists of a system of gymnastics and massage. The gymnastics consist of movements per-

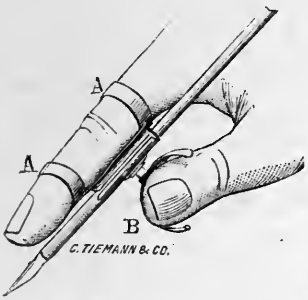


FIG. 228.—MATHIEU'S INSTRUMENT FOR WRITERS' CRAMP.

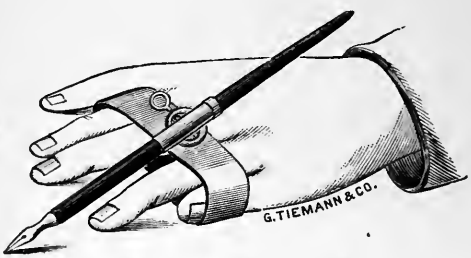


FIG. 229.—NUSSBAUM'S INSTRUMENT.

formed by the patient alone and movements executed with the co-operation of the operator. The first are performed by the patient during from twenty to thirty minutes, rarely for forty-five minutes. These movements consist of gymnastics of the fingers; extension, flexion, abduction, and adduction being performed, and the thumb being exercised separately. After this the four motions are executed at the wrist-joint, then extension and flexion of the forearm, and ultimately the arms themselves are exercised in the same manner and are to be lifted over the head. Each single exercise is to be performed from six to twelve times. After each motion a pause is to be observed. The opposed movements are to be carried out in the same manner, except that the operator must carefully resist their execution as though he were endeavoring to force the patient to perform a motion just the reverse of his intentions. Regularity of pressure is to be observed in this, so that the same amount of force is always used and so that the pressure does not

vary in intensity from moment to moment. The time to be devoted to these opposed movements should be the same as that for the unopposed ones. According to the intensity of the affection, the exercises must be repeated two or three times daily. The massage itself consists of two parts—nerve and muscle massage. The nerve massage is effleurage along the course of the nerve trunks, the median, ulnar, and radial, going upward to the axillary and cervical plexuses. This effleurage lasts about ten minutes. Following this is the muscle massage. This consists of pétrissage, beginning with the hand and ending at the shoulder. The duration is the same as that of the last movement. One sitting a day has always proved sufficient. Wolff, in addition, uses "a peculiar method of writing instruction" and employs rubber bands and rings in his manipulations. It must be added that one hears very little of the Wolff method at the present time.

Electricity ranks second to massage in the treatment of occupation neuroses. The high-tension faradic current with long coil has done good service in some of my cases. The galvanic current has been helpful also. It should be given daily. The anode is placed over the cervical spine and the cathode over the various muscular groups affected. A stabile current of five to ten milliamperes for from ten to fifteen minutes is given.

Lotions containing muriate of ammonium, liniments, hot and cold douches, the cautery, all have been recommended in professional neuroses. Tenotomy was once employed, but has been abandoned. Very little can be expected of drugs. The most trustworthy are atropine, strychnine, cannabis indica, the iodides and bromides, and cod-liver oil. It should be remembered that sometimes the disease is almost purely cerebral, and then an antineurasthenic treatment is called for. But in other cases, when the disorder is largely peripheral, the usual treatment for a low grade of myoneuritis must be employed.

CHAPTER XXVII.

PARALYSIS AGITANS (SHAKING PALSY, PARKINSON'S DISEASE).

Paralysis agitans is a chronic progressive disease, characterized by tremor, muscular rigidity and weakness, and by a peculiar attitude and gait, together with sensations of heat, pain, and restlessness.

Etiology.—It occurs oftenest between the ages of fifty and sixty, then between sixty and seventy and forty and fifty. In very rare instances it occurs in early life, but the genuine disease does not occur before puberty. Males are affected much oftener than females (five to three in seventy-eight American cases). It occurs in all classes of life, but oftener among those who incur exposures and endure hard labor. It is not a disease of vice and is not the result of alcoholism, syphilis, or sexual excess. Prolonged overwork and anxiety in middle life are very often predisposing causes. Heredity is a rare factor, but I have known hereditary family tremor to end in paralysis agitans. It appears to have some relation to rheumatism and especially to rheumatoid arthritis. It occurs oftenest in this city among the Irish, German, and Polish races (twenty Irish, thirteen Germans and Russians, the last mostly Hebrews).

The apparent exciting causes in the majority of cases are exposure to wet and cold, fright, injury, and acute mental suffering. An attack of rheumatism, a sudden severe muscular strain, and fevers are rare causes. The actual exciting cause is probably always an infection, just as in multiple sclerosis, paralysis agitans being the senescent counterpart of that disease.

Symptoms.—The disease is sometimes ushered in with an acute illness, or an attack of sciatica. It then develops slowly with some aching pains in the arm and a slight tremor in the fingers of one hand, oftener the left. This gradually extends and involves the foot of the same side, then the other side becomes affected. The neck, face, and tongue are rarely attacked, and then to a small extent. After or with the tremor there comes on a stiffness in the arms and legs, and indeed of the whole body. With this there is a general contracturing and shortening of all the flexor groups; so that the head and body are bent forward, the fingers are straight but are flexed

as a whole on the metacarpus, the forearms flexed on the arm, the trunk is flexed forward on the thighs, and the knees are slightly bent. The attitude gives the idea of extreme senility (Fig. 230). The gait is slow, the steps are short and shuffling; the patient has trouble in starting, stopping, and turning corners, owing to the slowness in initiating new movements in the voluntary muscles. When once started he may be unable to stop and has to run along. The speech early becomes affected. The voice is high-pitched, weak, and piping, or senile in quality. There is a slowness in getting out words or in starting a sentence, though after it is begun the words come rapidly. The condition is analogous to the hestitation in the gait.

Fig. 230.—Attitude in Paralysis Agitans (Curschmann).

Along with the other symptoms there are often, though not always, sensations of heat, burning, fever, and rarely of coldness. These sensations are felt most in the feet, legs, or arms diffusely. Often there is a general feeling of restlessness and nervousness. Aching pains and a sense of fatigue occur; neuralgic pains are more rare. There are always a peculiar redness and flush in the faces of the patients. Sometimes they sweat profusely. The temperature in the axilla is normal, on the skin it is sometimes increased (Peterson). The appetite is excellent, often abnormally great, and digestion is good. Visceral complications are rare. Muscular weakness comes on early; it slowly increases, but complete muscular paralysis does not occur. The disease ends in rigidity, which makes the patient as helpless as if paralyzed, but the muscles preserve considerable functional power to the last. The deep reflexes are present and not, as a rule, exaggerated; but exaggeration and even clonus occur in a small percentage of cases. As the disease progresses the tremor increases in extent, and continues without remission during all the waking hours; the limbs get more rigid; the patient becomes

bedridden and is finally carried off by exhaustion or some intercurrent illness (Fig. 231).

It will be seen that the dominant symptoms in paralysis agitans are:

1. Tremor.
2. Rigidity, progressively increasing.
3. Muscular weakness.
4. Sensory and vasomotor disturbances.

Further details must be given regarding these symptoms:

The *tremor* is at first rather fine, but later is coarse. It ranges from about 6 vibrations per second to 3.7. The average rapidity is 4 or 5 per second, which is about one-half the normal muscular

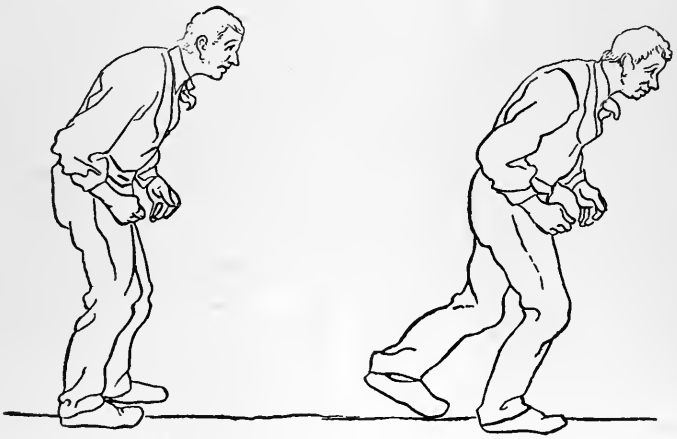


Fig. 231.—Attitude and Gait in Paralysis Agitans.

rhythm. But the chief characteristic of the tremor is that it continues when the hand or limb is at rest, while voluntary motion causes it to cease. As the hand rests on the knee it shakes; as it is moved the tremor stops. When held straight out there is no shaking for a moment, but it soon begins. A glass of water is carried safely to the lips. The patient can control the tremor for a moment, especially in the early stages of the disease. These facts about the tremor apply in ninety per cent of cases, but there are patients whose tremor is slight when the limb is at rest and is increased on voluntary effort. The hands are affected in a characteristic way. The fingers and thumb are slightly flexed and held about in the writing-position; the tremor moves the fingers and thumb as a whole, and they vibrate so that the one pats the other gently. Sometimes the tremor is one of alternate supination and pronation of the forearm. The neck and face muscles are not usually or ex-

tensively involved, the shaking of the head being generally the result of the general bodily tremor. Sometimes one sees a tremor of the lips or neck muscles. The tongue and eye muscles are practically never involved.

Rigidity.—The rigidity comes on early, and may be the first and even the only prominent symptom. It affects chiefly the

FIG. 232.—TERMINAL STAGE OF PARALYSIS AGITANS, showing rigidity and contractures.

flexors of the arms, head and trunk, and legs, producing a characteristic senile position. In rare cases the extensors of the neck are affected and the head is drawn back. Cramps occur, and there is always a sense of stiffness. The muscular movements are slow, especially the initiation of a movement. Once started, a motion may be quickly done. The gait is peculiar: the steps are short and shuffling; the patient may have difficulty in starting, but once started he goes along very well; or while walking there may be a sud-

den running forward. This is called "festination." Rarely there is a tendency to run backward or sideways. The facial muscles are stiffened and little used, so that the face has a peculiar expressionless look. The patient is often emotional, but the mind is not seriously affected. The urine is usually about normal, but contains an excess of phosphates. There may be polyuria and less often glycosuria.

Forms.—The unusual types of paralysis agitans are the hemiplegic or the monoplegic, the rigid type, and the retrocollic type. The only one of importance is the rigid type, in which there is practically no tremor.

Course and Duration.—The disease slowly but steadily progresses until a full development of symptoms occurs, when it may

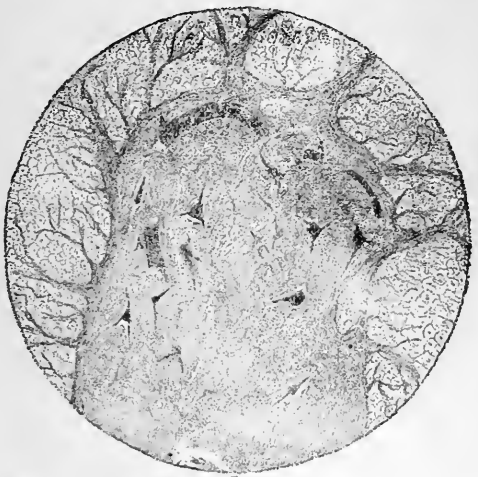


FIG. 233.—ANTERIOR HORN OF SPINAL CORD, showing dilated veins.

remain stationary. It takes about two years for the whole body to be affected, though this varies much. It lasts from three to twelve years or even more. In three cases of mine death occurred in three, six, and eleven years. Death is due to exhaustion and may be accompanied by mild delirium and fever.

The *diagnosis* must be made from senile tremor, multiple sclerosis, post-hemiplegic tremor, and wryneck affecting the extensors bilaterally (retrocollic spasm). Senile tremor occurs in the very old and affects the head first and most. In multiple sclerosis the tremor is more jerky and is a tremor of motion; there are nystagmus, syllabic speech, and often apoplectiform attacks, eye trouble, and paralyses.

Post-hemiplegic tremor is accompanied by a history of hemi-

plegia; there are paralysis and exaggerated reflexes and the disease is unilateral. In retrocollic spasm only the neck muscles and fron-

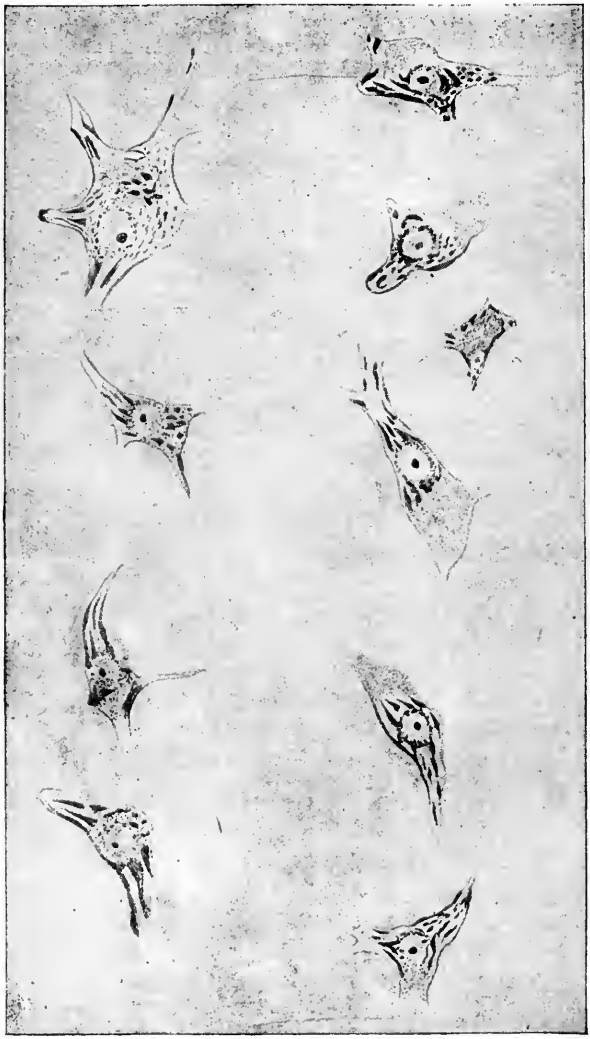


FIG. 234.—CELLS OF THE ANTERIOR HORN OF THE SPINAL CORD. The right row from a case of paralysis agitans, showing atrophy and pigmentation, the left row from a normal case.

talis are involved. The absence of exaggerated reflexes, the peculiar voice, gait, and attitude, and the sensations of heat and nervousness often help greatly in the diagnosis.

The *prognosis* is favorable as regards life; unfavorable as regards cure; and not very good as to bringing about a cessation of progress in the symptoms. The progress of the malady, however, can be delayed.

Pathological Anatomy and Pathology.—The post-mortem changes are not very marked, and are seen mostly in the spinal cord and medulla. There are congestion and dilatation of vessels in the gray matter, a diffuse increase of interstitial tissue, atrophy and pigmentation of cells (Figs. 233 and 234). The process is suggestive of a chronic interstitial inflammation with cell degeneration. It is probably a post-infectious process, with a toxin behind it. The cerebro-spinal motor neuron is the most at fault; and it seems as if the connections between its end brushes and the motor cells of the spinal cord were interfered with. Hence the peculiar "hold-ups," the rigidity and tremor of the disease.

Paralysis agitans is certainly not merely a premature senility, as some have taught.

Treatment.—The most important measure is rest, mental and physical, with plenty of fresh air. No special diet is indicated. Lukewarm baths and mild massage are agreeable and helpful. I know of no climatic cure. The galvanic current produces temporary relief; it should be given daily. Hyoscine hydrobromate, first used by Charcot and introduced into this country by Seguin, is of much temporary value in relieving the tremor (gr. $\frac{1}{100}$ increased). Codeine and morphine give the best permanent results. Quinine and mineral acids are of much service in relieving the vasomotor and sensory symptoms. I have used bromide of uranium (gr. $\frac{1}{30}$) with some apparently good results. Arsenic, Indian hemp, tinct. veratrum viride, salicin, and salicyate of sodium rank next in value. Nitrate of silver, conium, curare, bromides, atropine, phosphorus, cod-liver oil, iron, and picrotoxin have all been recommended. Bromide often helps the insomnia and restlessness. Extract of pituitary gland in doses of gr. xxx. to xl. daily quiets the system also.

Suspension is of some use in a minority of cases not too much advanced. The mind in paralysis agitans is sometimes in an emotional, almost hysterical, condition, and patients are easily made better for a time by some psychical influence. Hypnotism by means of fascination is said to be of use, but it has failed in my experience.

CHAPTER XXVIII.

TROPHIC AND VASOMOTOR DISORDERS.

PROGRESSIVE FACIAL HEMIATROPHY is a disease characterized by a progressive wasting of one side of the face.

Etiology.—It begins oftenest in the young between the ages of ten and twenty. Females are more affected.* There is in rare cases a hereditary history. Injury and infectious fevers sometimes start up the trouble. The left side is oftener attacked.

Symptoms.—The disease begins very gradually and shows itself

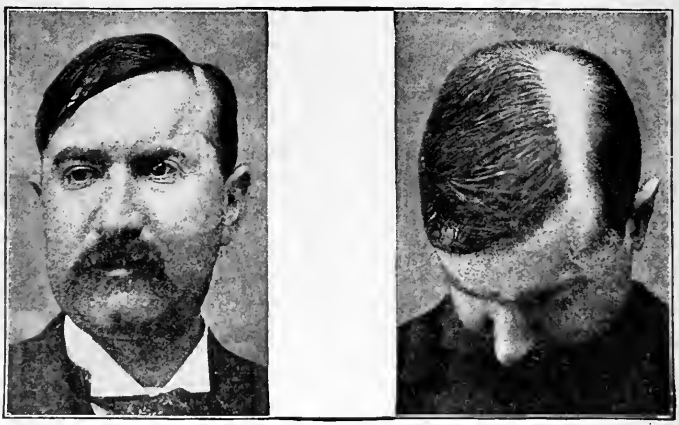


FIG. 235.—FACIAL HEMIATROPHY, EARLY STAGE, showing alopecia and osseous depressions.

first in patches. The skin gets thinner, there is loss of pigment, hairs fall out, and the areas may have a yellowish appearance. Sometimes the periosteum and bone are affected, and shallow depressions are formed which may be anæsthetic (Fig. 235). The subcutaneous tissue is most involved, the muscles suffer least, and there are no changes in electrical reaction. The muscles of mastication are usually spared. The bone undergoes general atrophy and the lower jaw may be reduced to two-thirds the normal size. The secretion of sebum ceases, but that of sweat may be increased. The

* About 100 cases have been reported. Among 5 seen by myself, 3 were in females, 2 in males. The disease in all cases, so far as could be found, began between the tenth and twentieth years.

temperature falls. There are a sinking in of the eye, narrowing of the lid, and dilatation of the pupil. There is sometimes pain and rarely anæsthesia. The tongue and other parts of the body may be involved. Spasmodic movements of the muscles of mastication have been noted (B. Sachs). Scleroderma sometimes appears on the face or hands.

The disease progresses rather rapidly at first, but finally comes to a standstill. It does not shorten life.

Pathology.—There has been found a degenerative neuritis involving the fibres of the trigeminus; its descending root and the substantia nigra were atrophied (Mendel).

The *diagnosis* is easy. Hemiplegia with atrophy in children, congenital asymmetry, and atrophy from gross lesions of the nerve are distinguished by their stationary character or the presence of severe pain.

Treatment. — There is no treatment known to be of service. Tonics, iodides, and electricity may be tried. Dercum has suggested resecting the trigeminal nerve.

Fig. 236. — Facial Hemihypertrophy Occurring in a Giant.

Progressive Facial Hemihypertrophy is an extremely rare condition, only eleven cases having been reported. It is usually congenital in origin, but may develop in connection with giantism, as in a case of my own (Fig. 236).

Acromegaly (Marie's Disease).

Acromegaly is a chronic dystrophy characterized by gradual enlargement of the hands, feet, head, and thorax, and by a dorso-cervical kyphosis. Though the disease was first described only ten years ago by P. Marie, the number of cases reported is rapidly increasing, and if one includes various abortive types it is not extremely rare.

Etiology.—It affects the two sexes nearly alike. It begins between the ages of eighteen and twenty-six; recently a congenital case has been reported. No hereditary influence or definite exciting cause is known. The patients are sometimes naturally endowed with large extremities.

Symptoms.—The disease begins with a gradual enlargement of the hands, feet, and head. In women there is amenorrhœa, in men sexual weakness; slight rheumatic pains, headaches, malaise, mental hebetude, anæmia, and general weakness are present. The skin is dry and there is polyuria.

The hypertrophy affects the soft parts as well as bones. In these latter there are periosteal thickening and hyperplasia, with the result of producing increase in width more than length. The arms are not much involved, nor is the shoulder girdle, except the clavicle. The lower jaw is much more involved than the cranium. The tongue, lips, and nose are enormously hypertrophied (Fig. 237). The thorax is enlarged antero-posteriorly and flattened. There is sometimes dulness over the sternum due to persistence of the thymus. The pelvis may be enlarged, but the hip and leg bones are gener-

Fig. 237.—The Face in Acromegaly (Curschman).

ally spared. The hands and feet undergo enormous hypertrophy (Fig. 238). The following are some of the measurements in the case that has come under my observation, reported by Adler, and in cases reported by Osborne and Packard:

Length of hand,	7.6 to 8¾	inches.
Length of foot,	11.7 to 12¼	"
Cranial circumference,	24 to 26½	"
Circumference of thorax,	44	"

The vision is sometimes impaired and there may be hemianopsia. The muscles may be at first hypertrophied, later atrophied. There are no paralyses and rarely any anæsthesiæ.

The disease runs a very chronic course, lasting ten or twenty years.

Pathology.—There has been found an enlargement of the pitu-

itary body in nearly all cases, and it is probable that the disease is due to disorder of its function. The attempts to place the disease in relation with a persistent thymus, sclerotic changes of the sympathetic, and disease of the thyroid, all of which conditions have been found, are unsuccessful. The disease must be regarded as a perversion of nutrition due to defective action of the pituitary gland.

The *diagnosis* must be made from congenital enlargements, from so-called giant growth which affects single members, and from oste-

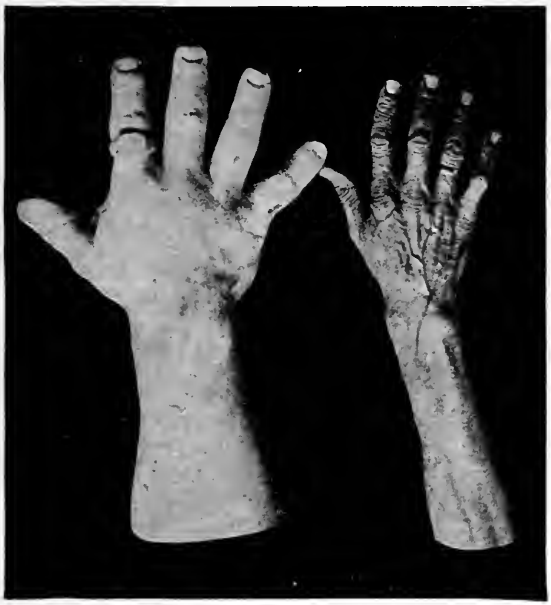


Fig. 238.—Normal Hand and Hand in Acromegaly.

itis deformans. In the latter disease it is the shafts of the long bones and the cranium, not the face, which are involved.

Pneumogenic osteo-arthropathy is the name given by Marie to a disease associated with pulmonary and pleuritic disease, and characterized by enlargement of the extremities and peculiar deformities of the terminal phalanges. The enlargements are not uniform. The tongue is not affected. The wrist and ankle bones are hypertrophied, the finger-tips are bulbous and spade shaped.

Prognosis.—Acromegaly is incurable, but it has been arrested, or at least has ceased to progress, and it may not greatly shorten life.

Treatment.—Cases have been reported in which iodide of potassium and arsenic have arrested the disease. In general, the treatment is only symptomatic, but feeding with pituitary gland in large doses (gr. xl.) should be tried.

Myxœdema.

Myxœdema is a disease of the thyroid gland, but its symptoms are so largely nervous that a brief description of it is justified here. It is a chronic disorder, due, as a rule, to an interstitial thyroiditis, and characterized by a solid œdema of the subcutaneous tissue, dry skin, loss of hair, subnormal temperature, mental dulness, and even insanity and idiocy. It has two forms—the congenital and infantile

Fig. 239.—A Case of Myxœdema in a Man aged Forty-Four Years (Murray).

—causing a condition known as cretinism; and an adult form constituting myxœdema proper.

It occurs most often between the ages of thirty and fifty, and oftener in women (seven to one). It is seen oftenest in temperate climates. Hereditary influence, alcoholism, and syphilis are not predisposing factors; lead poisoning may be a cause.

It begins slowly. The patient is languid and dull, and is unusually sensitive to cold. Voluntary movements are slow; the weight increases and a solid œdema which does not dent on pressure develops in the face and extremities. The skin gets dry and rough, the hair begins to fall, the temperature is subnormal, 1° to 2° F.

Mentally the patient is dull, forgetful, depressed, and in one-fifth of the cases melancholia, mania, or dementia develops. The muscles are weak, the gait is slow, the voice hoarse and monotonous. There is considerable anæmia and the heart is weak. The pulse is slow and the arterial tension low. Albuminuria is present in twenty per cent of cases, and hemorrhages may occur.

The pallor, œdema, loss of hair, and mental hebetude give to the face a characteristic expression (Fig. 239). The disease may run a course of six or seven years, the patient dying of cardiac weakness or some intercurrent malady.

The disorder is due in most cases to a chronic interstitial thyroiditis which usually causes atrophy of the gland. It may be produced by artificial removal of the thyroid. The result of this is a defective action of the thyroid, and a consequent poisoning of the system and deposit of mucin in the subcutaneous tissue especially.

The diagnosis is based upon the peculiar physiognomy due to the œdema pallor; the loss of hair, subnormal temperature, mental hebetude, and atrophied thyroid.

The prognosis is good if treatment is instituted.

The treatment consists in the administration of the thyroid extract in daily doses ranging from five to forty grains or even more. The results are most brilliant, and humanity owes much to Dr. George R. Murray, who first instituted it.

Cretinism.

Cretinism is a form of myxœdema due to absence, atrophy, or defective function of the thyroid gland, occurring congenitally or dur-

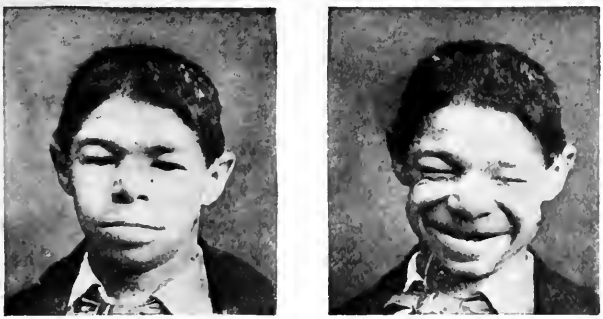


Fig. 240.—A Cretin Dwarf, aged Twenty (Leszynsky).

ing infantile life. The disease occurs endemically in parts of Europe, but only sporadically and happily with great rarity in America.

Hereditary and family influences are at work in endemic but not in sporadic cretinism. It develops either directly after or in the first three years of life, and shows itself in a stunted growth both of brain and body, most cretins being idiotic dwarfs. The general symptoms are much like those of myxœdema in adults plus the retarded growth of mind and body. The deposits of solid œdema cause peculiar deformities and lead to a characteristic physiognomy.

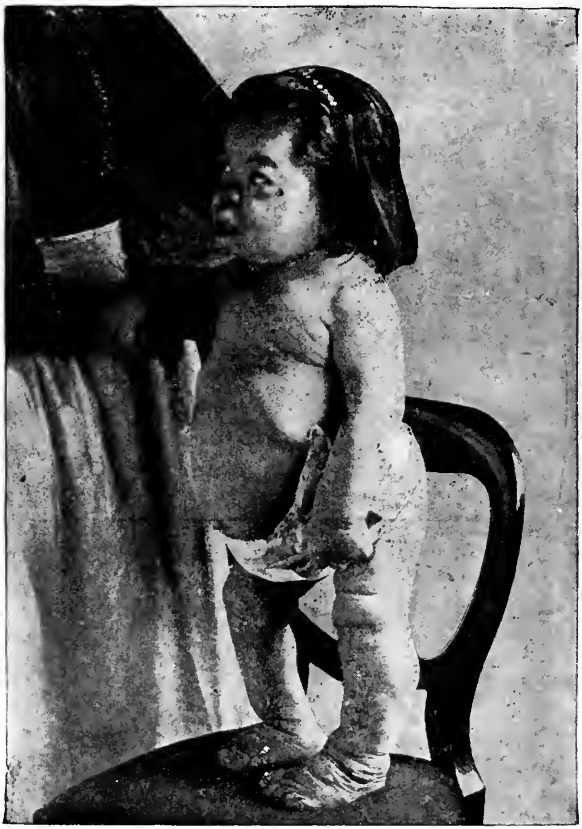


FIG. 241.—SPORADIC CRETINISM IN A PATIENT AGED TWENTY-EIGHT; HEIGHT, 34½ INCHES (Murray).

The mind is dull and placid, the muscles are weak, the abdomen is protuberant, the hands and feet are broad and thick; the patients are anæmic, the temperature is subnormal. The arrested bodily growth is such that on reaching adult age the stature may be only twenty-eight to thirty-three inches (Fig. 241).

Cretins usually die young, but some survive to the age of thirty or forty.

The treatment is the same as that of myxœdema. Here, too, if the case is seen before adolescence, brilliant results can be obtained.

Angio-Neurotic Œdema (Circumscribed Œdema).

Angio-neurotic œdema is a functional disorder characterized by the rather rapid appearance of circumscribed swellings upon different parts of the body, these swellings being due to disturbances of vasomotor innervation and not of an inflammatory character. The disease occurs oftenest in early adult life, the average age being from twenty to thirty, but it has been observed in young children and even in the aged. It occurs oftener in males than in females, except in this country, where the reverse ratio exists. Hereditary influence plays a part in some cases; the disease has been known to run in families. It occurs oftener in winter and oftener in the early morning hours. Exhausting occupations predispose to it. The exciting causes are sudden exposure to cold, slight traumatisms, fright, anxiety, grief, and the ingestion of certain kinds of food such as apples or fish. A peculiar form of this œdema seems to develop in connection with menstruation.

Symptoms.—The disease appears without much if any warning. In a few minutes or hours there develops a circumscribed swelling upon the face or arms or hands. This swelling varies in diameter from one-half inch to two or three inches. It may be dark reddish or rosy or it may be pale and waxy. It does not easily pit on pressure. There is sometimes a local rise, sometimes a fall in temperature. It is accompanied by sensations of tension and stiffness, scalding, burning, and sometimes itching, but there is no actual pain. The swelling is usually single, but it may be multiple. It is located most often upon the face, next upon the extremities, particularly the hands; next on the body, then in the larynx and throat, and then on the genitals. The swellings last from a few hours to two or three days. Between the attacks the patient feels well. They are apt to return at intervals of three or four weeks to several months. Sometimes they are brought out only by certain peculiar exciting causes, such as indigestion or mental anxieties or emotional disturbances. When the disease attacks the larynx or throat, serious symptoms of dyspnœa and suffocation may appear; surgical interference may even be called for, and death has been known to result. It has been thought that neurotic œdema may sometimes attack the stomach, producing symptoms of nausea, vomiting, and great gastro-intestinal distress, and an acute neurotic œdema possibly sometimes attacks the lungs. This, however, is unlikely, since the vasomotor innervation of the pulmonary blood-vessels is a very stable one.

The *pathology* of the disease is not known, except that it is unquestionably a disorder due primarily to disturbance in nerve innervation. The nerves affected are, farthermore, undoubtedly vasomotor nerves. The œdema is precisely similar to that which is associated with attacks of tic douloureux and migraine.

Diagnosis.—The symptoms of the disease are so peculiar that they are easily recognized. The spontaneous appearance of the œdema, its recurrence at certain intervals, and the absence of pain and evidences of inflammation are sufficient usually to enable us to recognize it. The giant urticaria is a disease resembling neurotic œdema and probably closely allied to it. The blue and the white œdema of hysterics differs in being persistent and associated with paralyses, anæsthesias, and contractures.

The *prognosis*, so far as cure is concerned, is not very good. The attacks, however, can be ameliorated, and the disease itself is not serious as regards life and the enjoyment of a fair degree of general health.

The *treatment* consists in the adoption of such measures as will give tone and stability to the nervous system; the use of cold baths, exercise, and massage is indicated. Internally mineral acids and strychnine may be of some value. Cascara, nux vomica, the salicylates, arsenic, quinine, and atropine are all drugs which have been recommended.

CHAPTER XXIX.

THE DISORDERS OF SLEEP.

INSOMNIA, HYPNOTISM, MORBID SOMNOLENCE, CATALEPSY, TRANCE, LETHARGY, THE SLEEPING SICKNESS.

SLEEP is a condition in which consciousness is normally lost and in which the whole body, but particularly the brain, enjoys functional rest, while constructive and nutritive activity goes on.

Physiology.—The most conspicuous phenomenon of sleep is the subsidence of the higher cerebral functions; yet other organs, notably the muscular system, also take part in the resting-process. The brain during sleep is slightly anæmic, the deficiency in blood being a part of, but not the cause of, the phenomenon. The remote cause of sleep is inherent in the nervous tissue itself, which follows the great rhythmical law, common to all living tissue, of rise and fall in its irritability. It is probable that the immediate cause of drowsiness is the exhaustion of the irritability of the cortical cells and the benumbing of them by the circulation of waste products in the blood. Many facts in the history of the pathology of the brain point to the existence of a sleep centre, which, being especially acted upon, tends to inhibit the consciousness and draw the mind into a somnolent state.

As sleep is only a function, we cannot speak of its diseases, but only of its disorders, and these really form but a part of the diseases of the brain or of general diseases. It is a matter of convenience, however, to discuss some of these separately.

Classification.—Custom has established the use of certain terms for the various disorders of sleep, and such terms must be for the most part adhered to. It will be proper, however, for the sake of completeness, to arrange the various disturbances we are to discuss in accordance with the modern methods of studying the pathological changes of bodily functions. We propose, therefore, the following classification, which indicates the various depressions, exaltations, and perversions of the functions of sleep:

I. State of normal sleep	*Hypnosis.*	Somnus.
II. States of absence of sleep.	*Ahypnosis.*	Insomnia.

III. States of perverted or artificial sleep. *Parahypnosis.*	Dreams, nightmare, night terrors, sleep-drunkenness, somnambulism, hypnotism.
IV. States of excessive or frequent drowsiness and sleep. *Hyperhypnosis.*	Morbid somnolence, paroxysmal sleep, epileptic sleeping-attacks, trance sleep, lethargy, sleeping-sickness of Africa.

I. Normal sleep varies much in accordance with age, sex, the individual, and, to a slight extent, with occupation, race, and climate. The infant sleeps fourteen or sixteen hours out of the twenty-four, the adult needs about eight hours, while the aged live healthfully with but six. Women need half an hour or an hour more than men. A few persons, generally men, need nine, ten, or even twelve hours of sleep daily; others require only six. Brain workers, as a class, take less sleep than laborers. Sleep is sounder and longer in cold climates and among northern races.

II. Insomnia is a term given to conditions in which persons simply suffer from insufficient and restless sleep or from entire absence of sleep for a long time. Such conditions result from a great variety of causes. It is my purpose to discuss only those forms in which the trouble is functional or nutritional, leaving out of consideration the symptomatic insomnia of organic brain disease and that occurring as the result of painful diseases.

An entire absence of the capacity to sleep occurs most often and typically at the onset or in the course of insanity. It is here a prominent and most distressing symptom. The length of time during which a person can live without any sleep is about the same as that during which he can go without food, viz., three weeks. Many hysterical, neurasthenic, or incipiently insane individuals will assert that they have not slept for weeks, but careful examination shows that they have at least been in a drowsy, somnolent condition, which is, in a measure, physiologically equivalent to sleep.

Etiology.—The cases in which persons can get only a troubled rest of a few hours are much more numerous. It is a disorder of the third, fourth, and fifth decades of life. Women are less liable to suffer from it than men, and the laboring classes less than those engaged in business or professional pursuits. A frequent symptom of neurasthenia is an imperfect, and especially an unresting, sleep. In gout and in the so-called latent gout, or lithæmia, insomnia is a frequent symptom. One of the few nervous symptoms of secondary syphilis is insomnia. Insomnia may develop as a bad nervous habit in persons who are neglectful of themselves. It occurs sometimes as an hereditary neurosis. I am acquainted with a family in which, for four generations, one or more of the members have suffered from

chronic insomnia throughout life. In anæmia and chlorosis there is often insomnia at night, combined with somnolence during the daytime. Disease of the heart and arteries may lead to insomnia, and under this head come the cases which occur in Bright's disease with tense arteries and anæmic brains. Disorders of the stomach lead to disturbed sleep oftener than to complete insomnia, and the liver, when inactive, causes somnolence rather than the contrary. The poison of malaria and the toxic agents of fever must be added to the list of causes of imperfect sleep.

It will be seen that the causes of chronic functional insomnia may be classed under the following heads:

1. Neurasthenic and vasomotor, including hereditary and habit insomnia.

2. Vascular and cardiac, including heart disease, arterial fibrosis, and general anæmia.

3. Auto-toxic or diathetic, including lithæmia, gout, and uræmia.

4. Toxic, including syphilis, lead, malaria, tobacco, and various drugs, such as coffee, tea, and cocoa.

In many cases there exists a combination of these causes.

Symptoms.—The forms and degrees of insomnia vary greatly. In children it is accompanied usually by much mental and physical disturbance. The patient is restless, excited, talkative, or querulous and irritable. The insomnic child is more ill than the insomnic adult. In neurasthenic insomnia there is a tumult of thoughts which prevent sleep, or sleep is superficial, unresting, and interrupted by dreams. In many cases of insanity insomnia is characterized by great motor restlessness. In old people insomnia is generally of the quiet kind.

Treatment.—As insomnia in all its phases is often a symptom of some general disorder, treatment of a curative kind must be directed to this. Anæmia, lithæmia, uræmia, malaria, and the other toxic influences must be removed by remedies adapted to these conditions. But besides constitutional treatment there is a symptomatic treatment which will be discussed here, premising, however, that while there are many sleep-producing medicines there *are no good drugs for insomnia.*

The older physicians in treating sleeplessness, used to depend largely on hyoscyamus, camphor, opium, and the fetid drugs, such as asafœtida, musk, and valerian. Hyoscyamus is still used. It is to be given in large doses, such as five or ten grains of the extract, or even more, and from ten to twenty drops of the fluid extract. The hydrobromate of hyoscine, in doses of gr. $\frac{1}{100}$ to gr. $\frac{1}{60}$ or

more, is one of the best forms. Hyoscine is indicated in the insomnia of the insane, especially in forms accompanied by motor activity. Chloral hydrate still holds its own as one of the surest of hypnotics. The dangers involved in its use have been somewhat exaggerated, though they are sufficiently real. Doses of gr. x. and gr. xv. are often quite large enough, but in alcoholic insomnia it may be given in twice the above amounts, guarded with ammonia and digitalis. Not a few persons find that chloral has bad effects. The patient awakes with a dull, heavy sensation in the head, slight headache, or gastric disturbance.

The various bromides are efficient and safe hypnotics if properly used. The immediate effect of them is simply sedative, and sleep is not produced unless very large doses are given. Some persons are even kept awake by average doses (gr. xv. to xx.). In insomnia, therefore, bromides are best prescribed in doses of gr. xv. three times a day. By the second evening sleep is generally secured. The bromide habit is rarely formed, and is in itself not so seriously injurious as the choloral or opium habit. The bromides alone are hardly strong enough hypnotics for alcoholic insomnia or the insomnia of insanity. I have found them to fail in the insomnia of the aged. Paraldehyde ranks close to chloral in its value as a hypnotic. In some persons it disturbs the stomach, but not in all, and it may be used as a hypnotic for months without its power being impaired. It is a disagreeable drug, and there is nothing, so far as I know, that palliates its offensiveness. I prefer, however, to prescribe it in ʒ i. doses poured upon a teaspoonful of powdered sugar. Doses of ʒ ss. are sufficient to cause sleep in many cases, and collapse may be caused by ʒ ij. given to weak patients. Urethane, in doses of a scruple or more, is a mild and agreeable hypnotic, but not so certain as paraldehyde. Amylene hydrate is a hypnotic of properties similar to those of paraldehyde, but less disagreeable. It is given in doses of about one drachm. Lupulin in large doses, gr. x. to gr. xx., is a good hypnotic. Sulphonal in doses of gr. x. to gr. xxx. given two or four hours before retiring is one of the best hypnotics. Chloral-amide is more agreeable and safer than chloral, though it acts practically in the same way. The dose is ℈ i. to ℈ ij. Trional in doses of gr. xv. is rather better than sulphonal.

Among the antispasmodics are several drugs which occasionally answer well in the insomnia due to nervous irritability. A drachm of the fluid extract of valerian or of spirits of lavender, for example, may be prescribed. In some forms of insomnias—perhaps best in those due to fever, or pain, or some rheumatic or gouty trouble—antipyrin in twenty-grain doses acts well. It is known that in

many cases of mild types of insomnia a dose of whiskey, brandy, or beer will put the patient to sleep.

Besides drugs, there are many hygienic or mechanical measures to which the physician may successfully resort—listening to monotonous noises, reading dull or heavy books, counting, or keeping before the fancy some blank or wearying picture—

> "A flock of sheep that leisurely pass by
> One after one; the sound of rain and bees
> Murmuring; the fall of rivers, winds and seas,
> Smooth fields, white sheets of water, and pure skies."

Mechanical remedies have nearly all for their purpose the withdrawal of the blood from the brain to the skin and abdominal viscera. Hot footbaths or warm general baths, cold douches down the spine, beating the limbs with rubber hammers, brisk exercise, a light meal, massage, all are at times efficient hypnotics. Persons who suffer from insomnia should sleep in cold rooms, the head should not be too high or very low, and in most cases they are better without late suppers, even though these be light. Mental work should be laid aside several hours before retiring, and the evening devoted to quiet conversation and reading or amusements that do not actively excite the nerves. Many persons live in good health though they sleep in the day and stay awake at night. Journalists and editors, whose work obliges them to go to bed in the early morning, often continue for years without impairment of physical vigor. Despite this, it is true that the best time for sleep is at night, and that the old maxim, "Early to bed," is a sound one. Yet it is not the early bird that gets the worm so much as the bird that has slept well. The human system requires a certain amount of sleep and should have it. The industrious and ambitious often try to train themselves to shorter hours, but though they may succeed for a time, nature will not be cheated out of her due and health suffers in the end. It is a widespread custom in some countries to take a short nap in the daytime, and the custom is a good one. America has not adopted it, but might do so with benefit to the health of her brain-working class. Many from childhood up do not get a sufficient amount of sleep.

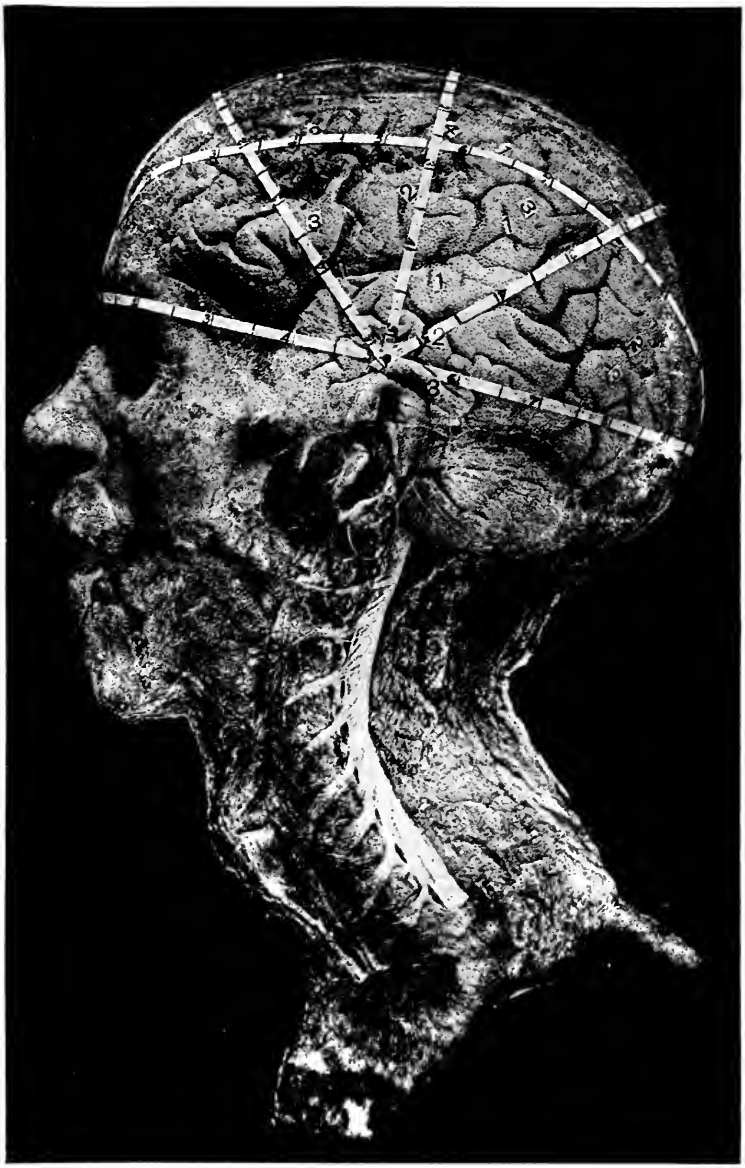
III. Perversions and Disturbances of Sleep.—Sleep is said to reach its deepest stage in from one to two hours after it begins. There is then after this a gradual lessening of the depth of sleep. Probably there are great variations in this rule, for many persons seem in soundest slumber several hours after falling asleep. But, at any rate, there are lighter stages of sleep at its inception and

toward its end. These are the favorite times for dreams, and at this period also there develop the peculiar phenomena of sleep drunkenness.

Dreams, Nightmare.—When sleep is perfect and profound, dreams afterward remembered do not occur. Dreaming is, therefore, a morbid symptom, although often of trivial significance, especially if it occurs at about the time of natural waking, when slumber is, in its physiological course, passing into the lighter stages. In sleep, no matter how light, the action of the regulating centre which directs thought, controls emotion, and exhibits itself in volition is suspended; the psychical mechanism, if excited to action at all, works without purpose, like a rudderless ship at sea. Ideas and emotions succeed each other by the laws of association, but are not properly correlated, and judgment and logical reasoning are gone. As a rule, dreams are made up of somewhat ordinary ideas and fancies incoherently associated, and shifting too rapidly to call up much feeling. When from some point in the body painful sensory excitations do produce disagreeable images, emotions of a most violent kind may be felt.

In the earlier stages of civilization, among primitive people, dreams were comparatively rare. When they did come with vividness they were regarded with importance, and often were considered visitations of spirits. Civilized man dreams more, but he has learned to treat his fancies with corresponding indifference. The attempts of scientific men to formulate laws regarding them have been productive of small results. Some diseases, however, cause, as a rule, dreams of a more or less peculiar kind. Thus heart disease is accompanied by dreams of impending death. Previous to attacks of cerebral hemorrhage patients have dreamed of experiencing some frightful calamity or of being cut in two. Intermittent fever is often announced by persistent dreams of a terrifying character. Hammond has collected a large number of what he terms prodromic dreams, all going to show that before recognizable signs of disease are present morbid dreams of various kinds may occur. Albers says: "Frightful dreams are signs of cerebral congestion. Dreams about fire are, in women, a sign of impending hemorrhage. Dreams about blood and red objects are signs of inflammatory conditions. Dreams of distorted forms are frequently a sign of abdominal obstructions and diseases of the liver."

Nightmare is a disorder incident to the hypohypnotic state, or that of incomplete sleep. It is one of those minor ills that are nearly always symptomatic of an irritation in some part of the body. The usual causes of it are some digestive disturbance (re-



COMPOSITE PHOTOGRAPH SHOWING RELATIONS OF CRANIAL SURFACE TO THE FISSURES AND CONVOLUTIONS (ALEC FRASER).

pletion) and cardiac disease. Persons of a nervous temperament are more subject to it; and there are individuals whom it makes suffer all their lives. The popular belief that sleeping on the back favors it is, in general, a correct one. When nightmare occurs in cardiac disease a certain position, semirecumbent or on the right side, must be maintained, or the painful fancies will awaken the patient. Healthy people can get sound sleep whether lying upon the back, the side, or the stomach; but light sleepers, and those with sensitive abdominal viscera, generally find that the position on the right side is the most comfortable and less provocative of unpleasant dreams. Prolonged mental or physical strain, excitement, and worry predispose to nightmare. Farinaceous foods, excessive use of strong liquors, coffee, and tobacco, all have a similar tendency. Nightmare occurs also in anæmia and malaria, and it may, in fine, be excited by morbid conditions in any part of the body. It sometimes occurs about the menstrual period in women. Its most common feature is a sense of suffocation or impending death.

Pavor nocturnus, or night-terrors, is a sleep disorder peculiar to children. It is allied to nightmare on the one hand and sleep-drunkenness on the other. It differs from the former condition in that the child continues to suffer from the distressing fancies for some time after he is awake. Night-terrors occur usually one or two hours after sleep has begun. The child wakes up screaming with fright, and perhaps runs about the room or seeks its parents for protection against some imagined harm. The disorder occurs in weakly, anæmic, nervous, or rheumatic children. It is due sometimes to lithæmia, or, as the older writers put it, rheumatism or gout of the brain. Digestive disturbances, worms, dentition, hereditary syphilis, mental strain, fright, and excitement are placed among the causes. It sometimes appears to be a paroxysmal neurosis allied to epilepsy. The disorder is usually harmless and the prognosis favorable.

Somnolentia, or sleep-drunkenness (*Schlaftrunken*), is a condition of incomplete sleep in which a part of the faculties is abnormally excited while the other is buried in repose. It is a kind of acted nightmare. The person affected is incoherent, excited, and often violent. He experiences the delusion of some impending danger, and while under it acts of violence have been committed. The condition is one of medico-legal importance, therefore, and has been discussed by writers on that science (Wharton and Stillé). Minor degrees of it are often noticed in children and in adults who are roused from a very profound sleep. It at times becomes a habit.

and a most annoying or dangerous one. The disorder in its severe form is fortunately very rare.

The treatment of morbid dreams, nightmare, and pavor nocturnus must be directed to a removal of the causes. Tonics, cardiac stimulants, laxatives, antirheumatics, attention to diet, are called for according to the condition of the patient. Change in surroundings is often necessary. Among symptomatic remedies the bromides are the best, except in lithæmia, when alkalies and salicylates may prove more serviceable. In somnolentia the patient should be prevented from getting into too profound sleep. He may be awakened once or twice during the night, or take a nap in the daytime. The head in sleeping should be raised high and the body not too heavily covered.

Somnambulism.—Somnambulism is a condition similar to hypnotism or the mesmeric state. In it volition is abolished and the mind acts automatically under the dominance of some single idea. It is an acted dream. Sight, hearing, and nearly all the avenues of sense are closed. The sleepwalker avoids obstacles and performs ordinary acts automatically, like an absent-minded man, which in reality he is. All those mechanisms which have been trained by constant repetition to act automatically, like that which preserves equilibrium, are active, and their powers may even be heightened, so that the somnambulist may walk along roofs or on dangerous roads and thread intricate passages without harm. The automatism of the somnambulist may continue for hours, until a journey has been performed or a task completed. He may carry out with success familiar mathematical calculations, write a letter, or work upon a picture, but he only follows along the lines established by constant iteration in his waking moments. He can originate nothing new. He is roused from his state with difficulty, and when out of it he remembers nothing of what has occurred.

Somnambulism usually arises from overeating. Sleeping with the head too low is another cause. Violent emotions act indirectly by disturbing digestion. The habit being once established, however, attacks occur without apparent cause. The disorder occurs oftenest in young people about the age of puberty, and it then attacks the sexes alike. Later in life women are more often affected. The disease is fostered sometimes at school by the attentions of the schoolmates. In most cases a condition of morbid sensitiveness underlies it. The patients are neurotic. Hereditary somnambulism has been observed. Its attacks have alternated with those of catalepsy. They are likely, after a time, to become periodical, occurring every week, fortnight, or month. The somnambulic state may

come upon a person in the daytime. It is then regarded as spontaneous trance, or hypnotism. It is not the case, however, that persons who are easily hypnotized are usually somnambulists, though the reverse may be true.

Somnambulism is a term that should include not only sleep walking but sleep talking.

The treatment of somnambulism is very much like that for sleep drunkenness. The patient's surroundings must be investigated, and unfavorable influences, such as may occur at school or from injudicious nurses, be removed. He should be prevented from sleeping too soundly, the head should be raised, the clothing light, the diet regulated. Remedies like iron, quinine, phosphorus, and cod-liver oil may be given. When the patient is discovered in the somnambulistic state he should not be awakened, or at least not until he is safely back in bed.

Hypnotism, Trance, Mesmerism.—*Major hypnotism* is a morbid mental state artificially produced and characterized by (1) perversion or suspension of consciousness; (2) abeyance of volition; (3) a condition of suggestibility leading the patient to yield readily to commands or external sense impressions; and (4) intense concentration of the mental faculties upon some idea or feeling.

Minor hypnotism is a state closely bordering on normal sleep in which there is a lowering of consciousness and a condition of suggestibility.

The proportion of persons of all ages found by Beannis to be hypnotizable was about eighteen or twenty per hundred. Children up to the age of fourteen are very susceptible. After the age of fifty-five susceptibility lessens. Men are almost as easily affected as women; but persons of a docile mind and those trained to some degree of mental discipline and capacity for submission, such as soldiers and artisans, are more sensitive. In this country the percentage of hypnotizable subjects is less than it is in Europe. Hysterical and insane persons are not very susceptible. Those who have been mesmerized once are more easily affected afterward, and may even pass into the state involuntarily.

Methods.—There are two ways of inducing hypnotism, the fixation method and the suggestive method. The former and older plan, devised by Braid, is to make the patient fix his eyes for five to ten minutes on some bright object at a distance of six or eight inches from the eyes and a little above the horizontal plane of vision. A modification of this is the fascination method of Luys, by which the patient is made to fix his eyes on revolving mirrors.

In the "suggestive method" devised by Liébault and Bernheim

the subject is placed in a chair in front of the operator. The operator then talks to the subject in a firm and confident voice, assuring him that he will go to sleep in a short time, telling him to make no resistance, that his sleeping will be natural, that nothing will be done to worry or fatigue him, that he will dream pleasant dreams, that he will wake up feeling better; then that he is feeling drowsy, his eyes are heavy, objects look confused, the lids are falling, they are closed—in a moment more the patient goes off to sleep. This requires some little time—five to fifteen minutes. It may fail the first time and succeed the second.

Hypnotic states may be self-induced by rigorously fixing the attention upon some object. The ecstatic states of the saints and the nirvana of the Buddhists are forms of hypnotism; so also are the trance states in which some clairvoyants and spiritualistic preachers place themselves; this same curious phenomenon is at the bottom of the so-called "mind-healing" science, and it enters into rational therapeutics and orthodox religion. The capacity of the human mind for hypnotism or semihypnotic states is, therefore, a most curious and important fact.

Symptoms of the Major Form.—The person who has been hypnotized at first sits or lies quietly in the position he has assumed during the manipulations of the operator. No notable physiological changes occur, as, for example, in the pulse, respiration, temperature, pupils, skin, etc. Some increase in the cerebral blood supply, however, is said to be present. The patient will now respond automatically to any outside command or will be dominated by any idea which is suggested to him. He will talk, or walk, or run, or gesticulate, assume expressions of fright, anger, or joy, entirely in accordance with the command given. Apart from these commands he is entirely dead to the outside world. He hears, sees, smells, tastes, and feels nothing. He can be burned, cut, or injured without showing any signs of feeling. At a suggestion he may be made cataleptic, somnambulic, or paralytic. This state is termed *somnambulistic trance.* If left to himself, he gradually sinks into a deep sleep, from which he can with difficulty be roused. After a time, rarely more than one or two hours, he awakes as from ordinary slumber. This latter state is called trance coma, or *lethargic hypnotism.* The attempts of the Charcot school to divide hypnotic phenomena into three forms, the somnambulic, cataleptic, and lethargic, are hardly successful. Sensitive subjects can be thrown at once into lethargy, catalepsy, or somnambulic states at the command of the operator.

The phenomena of hypnotism depend upon the wonderful sensitiveness and quickness of the subject in responding involuntarily,

with all his nervous energy, to outside suggestion. Dishonest persons may learn the latter trick and thus simulate the hypnotic state. Travelling mesmerizers utilize such persons largely; hence no confidence can be placed in the phenomena exhibited by them.

Minor hypnotism is produced by the "suggestive method" of hypnotizing. By this latter plan patients are thrown into various degrees of the hypnotic state from slight drowsiness to lethargy, but they are not somnambulic, and do not become cataleptic or anæsthetic.

Patients naturally come out of the mesmeric state through the channel of deep sleep or lethargy. Ordinarily they are dehypnotized by word of command, or by a pass of the hand, or any impression which the patient expects to be used for the purpose.

Hypnotized persons have been observed to have a diminution in the spinal reflexes and a muscular hyperexcitability. They sometimes show a most extraordinary exaltation of visual, auditory, or other special sense.

Pathology.—The underlying changes of the hypnotic condition are unknown and will probably long remain so. Hypnotism is no doubt associated with changes in the vascularity of different parts of the brain and with rapid breaking down of nerve tissue. Animals constantly subjected to hypnotic influence become demented (Harting, Milne-Edwards). The state of major hypnotism is probably pathological. It is a neurosis. Minor hypnotic states are but slightly removed from the normal, and their production is not injurious.

Diagnosis.—As hypnotic states may be imitated and as injuries or crimes may be done in this state, it is very important to be able accurately to distinguish it. Since the phenomena are all subjective, this is very difficult. The methods of value are these: 1. Careful examination of the general phenomena by experts while the subject is in the alleged hypnotic state. 2. Testing the muscular hyperexcitability by percussing motor points. 3. Tests of alleged anæsthesia by sudden burning, or pinching, or injuring the subject. 4. Tests of the tetanic muscular rigidity by the revolving tambour. In the hypnotic state the hand may be extended and held with perfect steadiness, while in conscious states a tremor soon appears. 5. Tests with glasses and other apparatus may be made to determine alleged anæsthesiæ of the special senses.

Therapeutics.—The practice of using major hypnotization is injurious, tending to exhaust the nervous force and weaken the will. It should be done only with the greatest care. Its utility in therapeutics I greatly doubt. It may relieve symptoms in the hysterical

for a time, but it cannot be of permanent benefit and is likely to lead to actual harm.

The induction of minor hypnotic states by suggestion is not harmful if carefully and moderately employed. Its practical results, however, are not great, and the method is tedious, uncertain, and sometimes ridiculous. It has its value in pedagogy, among children, in neurasthenia and in morbid habits. The general popularization of hypnotism by means of mind cures, Christian science, etc., accomplishes its results at the expense of mental demoralization; and faith-healing institutes are pernicious elements in society.

Morbid Drowsiness.—This is a very common symptom, which may be due to any one of the following causes: 1. Old age, when there is a weakened heart or diseased arteries, with cerebral malnutrition. 2. The diseased vascular conditions which precede cerebral hemorrhage. 3. The cerebral malnutrition occurring before or during certain forms of insanity. 4. Various toxæmiæ, *e.g.*, malarial, uræmic, cholæmic, and syphilitic. 5. Dyspepsia and gastric repletion. 6. Diabetes. 7. Obesity. 8. Insolation. 9. Cerebral anæmia. 10. Exhausting diseases. 11. Concussion of the brain. 12. Climatic conditions, cold, etc.

A very common cause of drowsiness is dyspepsia attended by some torpidity of the liver, the condition popularly known as "biliousness." Another frequent cause is malarial infection, which perhaps acts indirectly by impairing the functional activity of the liver. Drowsiness from these causes oftenest comes on in the afternoon. Anæmia is attended by drowsiness during the day, while there is often insomnia at night. Syphilis is more likely to cause insomnia, but in its third stage somnolent conditions may be produced which are of serious significance. Drowsiness occurs from the effects of severe cold. It sometimes develops when persons change their surroundings, especially on going to the seashore, for low levels and a high degree of atmospheric pressure seem to promote sleep. The drowsy state that sometimes follows concussion of the brain is a familiar phenomenon. Some persons, no doubt, acquire the habit of drowsiness. At first the trouble may have been induced by indigestion, "biliousness," or malarial infection, but it persists after the cause is removed. Such persons can hardly sit through a lecture, a church service, or any exercise requiring quiet and attention. As the morbid drowsiness here described is only symptomatic, its treatment need not be discussed. Such remedies as coca, coffee, tea, atropine, glonoin, do not produce results equal to expectations.

Morbidly Deep Sleep.—Certain persons, when they sleep, pass

into an almost lethargic slumber. Persons who sleep in this way often sleep a longer time than normal. They are awakened with difficulty, and then suffer with headache or disagreeable sensations throughout the day. The symptom may be a prodroma of insanity. Instances in which persons retire at the usual hour, but can with great difficulty be roused in time for the ordinary duties of the day, are not rare. Some of these are illustrations of the vice of indolence, but in other cases there is an absolute need of nine, ten, or even fourteen hours of sleep.

This disorder of sleep is most liable to occur in the young and in those of nervous temperament. It often seems to be a congenital condition, for which nothing can be done. In other cases it results from overfeeding and indolent habits. Treatment is much the same as that indicated for sleep-drunkenness and somnambulism.

Paroxysmal Sleep, Narcolepsy, Sleep Epilepsy.—It sometimes happens that persons suffer from sudden attacks of unconquerable drowsiness; they fall off into slumber despite every effort of the will. These are more than drowy sensations, for sleep, or a state resembling it, cannot be kept off. Some of these cases are of a purely nervous character, *i.e.*, the trouble is not due to a humoral poison or to organic disease, but to a paroxysmal change in the nervous centres of a vascular or chemical character, causing sleep. It may be that the patient is epileptic and the sleep seizure takes the place of the ordinary epileptic spasms.

Cases of epileptic sleep, or narcolepsy, and allied forms are not of frequent occurrence. Females are rather more often affected than males, and the susceptible age is from fifteen to forty. The disorder is brought on sometimes by fright, overstrain, and humoral poisons acting on a predisposed nervous system.

The course is chronic and relief is not always obtained. It should be remembered that syphilis, malaria, or anæmia, and indigestion may be elements in the trouble which are important, if not fundamental. Bromides in small doses are often useful factors in treatment. Change of occupation, of mode of life, or of climate may be essential to a cure.

Catalepsy, Trance, Lethargy.—Most of the so-called cases of prolonged sleep, lasting for days or weeks, are cases of spontaneously developed mesmeric sleep in hysterical women, or cases of incipent insanity (katatonia or stuporous melancholia). The phenomena in these cases may take the form of *catalepsy*, with waxy rigidity of the limbs, or *lethargy*. In cataleptic states the limbs may be placed in various positions and will remain there for several

minutes (Fig. 242). In lethargy or trance states the patient may be plunged into a deep and prolonged unconsciousness, lasting from one day to several years. These are the "sleeping girls" of the newspapers. Others are persons of a too ready susceptibility to mesmeric suggestion, who get into a morbid habit of going into mesmeric sleep spontaneously. In these states there may be a lowering of bodily temperature, slowing of respiratory and heart action, and excessive sluggishness of the action of the bowels. The patients can hear and may respond to suggestions, but they are apparently insensible to painful impressions and do not appear to smell, taste, hear, or see. The eyes are closed and turned upward, and the

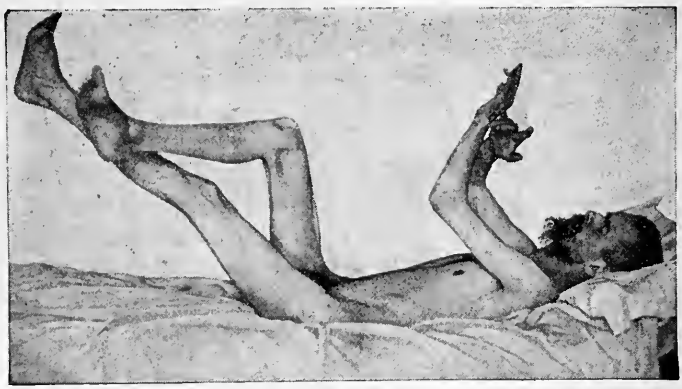


Fig. 242.—A Case of Catalepsy.

pupils contracted as in normal sleep. Many variations, however, occur in the physiological phenomena of these states.

The duration of the attacks of trance lethargy is from a few hours to ten years. Ordinarily, however, profound trance sleep lasts not more than a few days, while those cases in which the sleep is from mesmeric suggestion lasts but a few hours.

The katatonic patients after a few weeks or months gradually awake, become excited, and then pass into a condition of dementia or into catalepsy again.

Morbid Sleep from Organic Disease.—Prolonged and excessive sleep occurs as the result of syphilis of the brain, brain tumors, and the degenerative changes in old age and insanity. Morbid somnolence and stupor are not very frequent in cerebral syphilis, but are quite characteristic. The patient in some cases lies or sits all day in a semisoporous state; in other cases he walks about, but continually sleeps at his task. This state of partial sleep may pass off or end in complete stupor (Wood). It does not necessarily signify a

serious issue, even though it last for weeks. Somnolence or sleep is a rare symptom in cases of cerebral tumors other than syphilitic. Conditions of drowsiness or stupor have been noted especially in tumors of the basal ganglia and third ventricle.

Organic diseases of the brain tend to produce conditions of mental weakness, hebetude, or comatose states, rather than anything allied to sleep.

The Sleeping-Sickness, Sleeping-Dropsy, Maladie du Sommeil.—This is a peculiar disorder, apparently infectious in character, which occurs among the negroes of the western coast of Africa. The disease has been transported to other regions, but is endemic only in Africa. It begins gradually with some headache and malaise. Soon there is felt a drowsiness after meals. This increases until the patient lies for nearly the whole time in a stupor. When awake he is dull and apathetic. There seems to be no fever, and the temperature may even be subnormal; the pulse, too, is not rapid; the skin is dry, the tongue moist but coated, the bowels are regular. The eyes become congested and prominent. The cervical glands are enlarged. The disease ends in coma and finally death. Recovery rarely occurs. Sometimes the course of the disease is more violent, and toward the end there are epileptic convulsions and muscular tremors. Autopsies have revealed no definite pathological changes.

Accidents of Sleep.—Owing to the fact that sleep is a resting state of the organism, and that many of its functions are lowered, or their cerebral control lessened, peculiar crises, or physiological and pathological disturbances of nervous equilibrium, occur. Attacks of gout, of asthma, and of pulmonary hemorrhage are most liable to occur during the early morning hours. Deaths and suicides occur oftener in the forenoon, but births oftener at night. Epileptic and eclamptic attacks occur with much frequency at night. Involuntary emissions of spermatic fluid, orgasmic crises, and incontinence of urine are among the pathological incidents of sleep.

Disorders of the Prædormitium.—Sudden attacks of starting of the whole body, shock-like in character, accompanied by peculiar feeling in the head or occiput, not infrequently attack persons as they are dropping off to sleep. They are of slight significance.

CHAPTER XXX.

CRANIO-CEREBRAL TOPOGRAPHY.

The object of cranio-cerebral topography is to map out upon the scalp the underlying fissures, convolutions, and other parts of the brain. As this is for purposes of surgical operations, the mapping is done upon the shaved aseptic scalp with a soft anilin pencil dipped in strong carbolic solution (1 to 4), or with a brush and carbolized tincture of iodine. The only instruments needed are a steel tape measure and an instrument of nickel-plated soft iron. This consists of a flat strip 25 cm. long and 1 cm. wide. From its middle there branches a second strip 10 cm. long making an angle of 67° with the longer strip.* Practically, the principal points to be determined are the position of the longitudinal, Rolandic, Sylvian, and parieto-occipital fissures and the lower outline of the brain.

The measurements are based chiefly upon the known relations of certain landmarks on the skull to the parts beneath. These landmarks are the glabella, bregma, lambda, stephanion, asterion, and pterion, which are points at the junction of the various sutures with each other and with certain ridges or protuberances. Their position is shown in the cut (Fig. 240) except that of the glabella or prominence just above the nasofrontal suture. The inion is identical with the occipital protuberance.

The following rules are based upon the observations of Heftler, Thane, Reid, Horsley, Fraser, and myself:

I. The longitudinal fissure. This corresponds with the naso-occiptal arc.

II. The fissure of Rolando. Measure the distance from the glabella to the inion; find 55.7 per cent of this distance, and the figures obtained will indicate the distance of the upper end of the fissure of Rolando from the glabella. It should be about 48 mm. behind the bregma in male adults, 45 mm. in women, 30 to 42 mm. in infants and young children respectively.

The fissure runs downward and forward for a distance of about 10 cm. measured on the scalp, the real length being about 8.5 cm.

* Special instruments called cyrtometers have been devised by Wilson and Horsley, but are not necessary.

The fissure makes an angle of about 67° with the anterior part of the longitudinal fissure. This direction is determined by the instrument above described or by the cyrtometer. The lower third of it is more vertical, and the lower end is 25 to 30 mm. behind the coronal suture. A line from the stephanion to the upper part of the asterion should about pass through it. The fissure is shorter in children.

III. The fissure of Sylvius runs nearly horizontally, and lies either under or a little above the uppermost part of the parieto-squamous suture. *This suture*, the *external orbital process*, and the *parietal eminence* are the guiding landmarks by help of which the

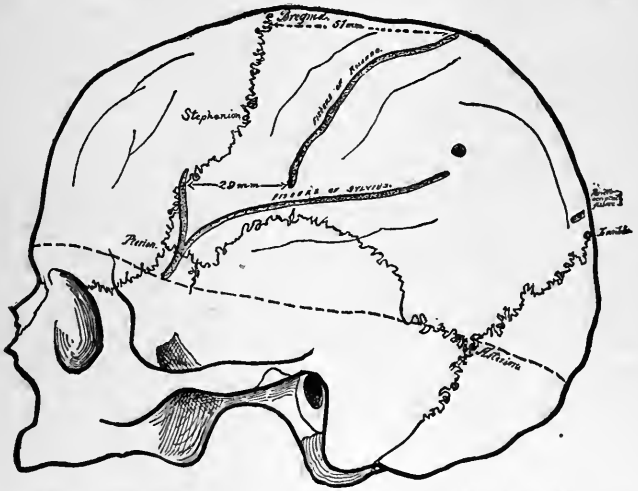


FIG. 243.—SHOWING THE POSITION OF THE BONY POINTS ON THE CRANIUM, THE SUTURES, AND THE PRINCIPAL UNDERLYING FISSURES, ALSO THE BASAL OUTLINE OF THE BRAIN.

surgeon can often operate without marking down lines on the scalp. In children the fissure is sometimes higher and more oblique.

To outline it, draw a vertical line from the stephanion to the middle of the zygoma. Draw a horizontal line from the external angular process to the highest part of the squamous suture; continue this back, gradually curving it up till it reaches the parietal eminence. The junction of the two lines will be at the beginning of the fissure of Sylvius. The vertical line indicates nearly the position of the ascending or vertical branch of the fissure, which is, however, directed a little more forward, and is about 2.5 cm. (1 inch) in length. The posterior part of the line indicates the position of the posterior branch of the fissure. Reid's method of finding the fissure of Sylvius is to "draw a line from a point 1¼ inches

behind the external angular process to a point ¾ inch below the parietal eminence. The ascending branch starts from a point ¾ inch back from the anterior end of this line, and 2 inches (5 cm.) back of the external angular process."

IV. To outline the parieto-occipital fissure, find the lambda, mark a point 3 mm. anterior to it, draw a line through this at right angles to the longitudinal fissure, extending about 2.5 cm. (1 inch) on each side of the median line. This marks the position of the fissure. If the lambda cannot be felt, its position may be found by measuring the naso-occipital arc and taking 22.8 per cent of it. This indicates the distance of the lambda from the inion or external occipital protuberance. The average distance in male adults is 7.42 cm. (2⅞ inches). It is greater in women than in men by a little over a millimetre.

V. To outline the frontal lobes: The anterior end of the frontal lobes reaches to a point determined by the thickness of the frontal bone. This ranges from 2 to 8 or more mm. (1/12 to ⅓ inch). The floor of the anterior fossa reaches in front to a level a little above the supra-orbital margin (16 mm., ¾ inch—Heftler). It slopes down and backward, its posterior limit being indicated by the lower end of the coronal suture.

VI. To outline the temporal lobe and the lower border of the cerebrum: The temporal lobe is limited above by the fissure of Sylvius, below by the contour line of the lower border of the cerebrum. This latter corresponds to a line drawn from a point slightly (about 12 mm.) above the zygoma and the external auditory meatus to the asterion, and continued on along the superior occipital curve to the inion. The anterior border of the lobe corresponds to the posterior border of the orbital process of the malar bone.

The temporal lobe is about 4 cm. (1⅝ inches) wide at the external auditory meatus. A trephine, as Bergmann states, placed half an inch above the meatus would enter the lower part of the lobe. The middle of the lobe is in a vertical line from the posterior border of the mastoid process. A line from the upper end of the fissure of Rolando to the point of the process would pass through this important sensory area (Fig. 243; see also p. 412).

VII. To find the position of the central ganglia, viz., corpus striatum and optic thalamus, draw a line from the upper end of the fissure of Rolando to the asterion, practically a vertical line. This limits the optic thalamus posteriorly. A vertical line parallel to the first, a little in front of the beginning of the fissure of Sylvius, limits the corpus striatum anteriorly. A horizontal plane 45 mm. (1¾ inches) below the surface of the scalp at the bregma limits

the ganglia superiorly. The ganglia lie about 35 mm. ($1\frac{1}{8}$ inches) below the superior convex surface of the brain (Féré).

VIII. To reach the lateral ventricles: A number of routes may be taken. The lateral is recommended by Keen. Mark a point $1\frac{1}{4}$ inches behind the external auditory meatus and $1\frac{1}{4}$ inches above a base line made by drawing a line through the lower border of the orbit and the external auditory meatus. Trephine at this point and plunge the director into the brain in the direction of a

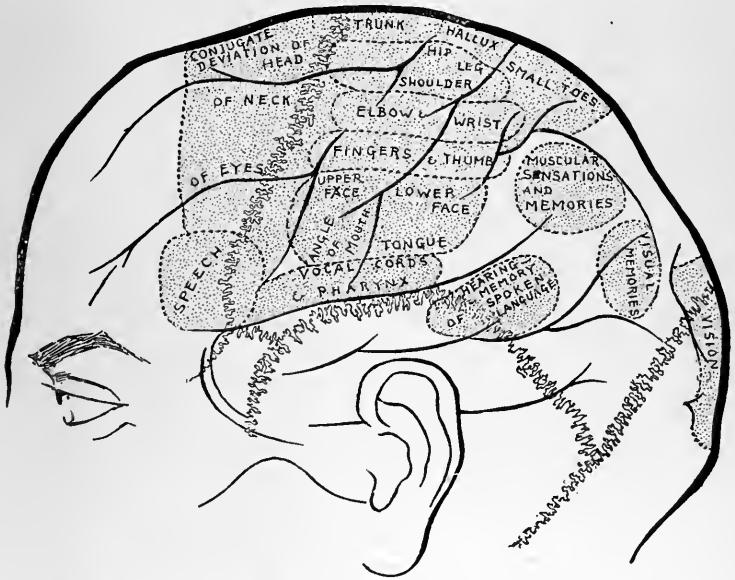


FIG. 244.—SHOWING THE RELATIVE POSITION OF THE FISSURES AND CORTICAL CENTRES OF THE BRAIN.

point $2\frac{1}{2}$ to 3 inches vertically above the opposite external meatus. The ventricle lies at a depth of 2 to $2\frac{1}{4}$ inches (5 to 5.7 cm.).

Mr. Alec Fraser has devised a way of mapping out the fissures by means of a series of composite photographs, so taken as to show the relation of the underlying parts to certain tapes tacked upon the skull. One of his figures is reproduced here (Plate III.).

In applying this method the surgeon tacks the tapes on the shaved scalp. Then looking at the diagram, he finds where the point in the brain is that he wishes to reach and notes its relation to the median lateral or circumferential tapes. Then as the circumference of the illustrated head is to the circumference of the living one, so is the position of the area on the tapes in the illustrated

head to the desired position of the same area in the living one. The illustration is a composite of several adult heads varying in circumference from 20¼ to 23½ inches.

The tapes are divided into inches and half-inches. The primary tape is the circumferential passing horizontally round the vault of the head (on the shaven scalp) from the root of the nose (glabella) to the maximum occipital point, which is about one inch above the inion. The tape which thus entirely surrounds the head is divided into four equal parts. The points where the division is made are at the anterior and posterior poles and midway on each lateral half of the tape. From these lateral mid-points a tape is run vertically over the top of the head, and other tapes are run anteriorly and posteriorly half-way between the point where the transverse tape crosses the sagittal suture and the anterior and posterior poles. Another longitudinal tape is run from the anterior to the posterior pole half-way between the sagittal suture and the circumferential tape.

APPENDIX.

ON NEUROLOGICAL THERAPEUTICS.

It has seemed best to describe, in a separate chapter, some of the technical details of the treatment of nervous diseases.

Hydrotherapy.

The apparatus needed for applying water in therapeutics consists of a room at least sixteen by twenty feet, well ventilated, with waterproof floor and walls. The floor must be made so that it will drain away the water flowing upon it. The room should contain a stationary bathtub, a shower or rain bath, an apparatus for giving hot and cold douches at various pressures, a hot box in which a patient can take a hot bath with the head exposed, a few foot baths, ice bags and ice caps. The most important point in detail is the douche apparatus, which should be supplied with all the improvements for regulating the heat, pressure, and impact of the stream. The stationary bathtub should be of the largest possible size, rather shallow, and placed low down so that patients can move their limbs easily and get exercise while in it. Among the accessories may be a "vaporium," devised by Dr. Percy Wilde for local application of heat or cold. It consists of a double copper cover, made in two sizes, one suitable for a single limb, the other for both extremities, the abdomen, or thorax. Boiling water is poured into the upper part (at *B*), and permitted to escape by two outlet pipes (*C*) at the bottom. This apparatus has been greatly elaborated recently, so that temperatures of over 200° F. are reached and applied locally.

While all the foregoing things are needed in an institution and should be placed in every hospital and asylum, most of the hydrotherapeutic procedures can be carried out fairly well with a stationary tub and a shower above it. A cheap hot box with a hole in the top and a lamp for heating below can be easily added if needed. The common forms of hydrotherapy prescribed by myself are the hot

box and Charcot or Scottish douches, the cold sitz bath, the drip sheet, and wet pack.

Special Applications of Hydrotherapy—*Epilepsy.*—Most patients should be made to take cold showers or sponge baths with a vigorous rub-off daily in the morning. Once a week they should take a hot bath. A cold plunge into a tub full of wat r is a good substitute for the shower. Persons who have neither shower nor tub should get a foot tub and a large sponge. Standing in the tub with a basin of cold water before them, they should fill the sponge and let the water trickle over the head, back, and body generally. This is done for two or three minutes, then the patient rubs himself down. The foot tub may have little warm water in it at first.

With delicate persons hydrotherapeutic treatment must be begun carefully. The first applications should be dry, warm flannel packs, then wet packs gradually made cold. Finally, cold drip sheets, showers, plunges, and rubbing may be used.

A good method for a fairly robust person is that originally described by Fleury. This consists in giving simultaneously the rain shower and the jet. The patient standing in the shower receives a jet of water on the posterior surface of the body for fifteen seconds; then the jet alone for fifteen seconds; finally the jet alone on the anterior surface of the body for thirty seconds.

In *neurasthenia* of adult life in men the patient should take the cold shower or plunge daily. In addition to this or in place of it, if needed, I strongly advise the Scottish douche thrice weekly or oftener. This may be preceded by a short hot bath. In neurasthenia of adolescence the same treatment is often indicated, but it must be applied more carefully, as many patients do not react well at first. They may require wet packs and lukewarm baths as sedatives for a time. Nervous women almost always do better with a preliminary course of dry and wet packs, followed later by showers and douches. Thus I prescribe first a hot flannel pack for one half to one hour (see p. 64); next day a wet pack, using a hot sheet; next day a cooler sheet, until in one or two weeks the patient takes a cold wet pack thrice weekly. If this, however, does not lessen irritability and quiet the patient, I prescribe a hot-air bath followed by a shower or douche.

Insomnia.—The lukewarm bath at a temperature of 90° to 96° F. is often efficacious. It should be taken at night before retiring, and should last from fifteen minutes to three-quarters of an hour. A cold cloth may be laid on the patient's head. A simpler method of inducing sleep is to make the patient thrust the feet into a basin of cold water, 40° F. to 50° F. The legs are sponged up to the

knees. This is done for fifteen or thirty seconds, then the feet are taken out and briskly rubbed. The most efficacious measure often is the *hot* wet pack. The pack is taken like a cold pack, only the sheet is wrung out in hot water. The patient lies upon this, and the sheet and then the blanket are folded about him. The duration is an hour or all night. In the rest-cure treatment a drip sheet is used (see Partial Rest-Cure, p. 605).

Headache of a congestive character is helped by cold foot baths or cold douches to the feet; or a bath at 60° F., which should last ten or twenty minutes and be accompanied by friction to the feet and legs. In anæmic headaches the head should be wrapped in thin linen bandages wrung out in very cold water and covered with a few layers of flannel. After they are removed the head is to be rubbed dry and covered with a dry cloth.

In *hysteria* much the same kind of treatment is indicated as in neurasthenia. When there is much excitement the wet packs are indicated. In most of the major forms a half-bath at 60° with cold affusions, or the shower, with Charcot or Scottish douche, should be given. The ordinary treatment at the Montefiore Home consists of:

Cold affusions while standing in warm water, or a hot-air bath followed by rain bath for thirty seconds at 85°, daily reduced until 60° is reached, this to be followed by a spray douche for five seconds at 65° or jet douche for three seconds at 65° to 55°. The douche is reduced gradually to 50° or less, increasing the pressure from two pounds to thirty (Baruch).

When there is spinal irritation the filiform douche may be used as a counter-irritant, or the shower at 65° to 85°.

In *locomotor ataxia* different patients get relief from different forms of treatment. The very painful and hyperæsthetic cases are not in my experience much helped by hydrotherapy. One may try, however, lukewarm baths, 85° F. to 95° F., for ten to twenty minutes, with or without pine-needle extract. For leg pains, hot-air baths to the legs alone, followed by affusions at 60° to 70°, are recommended. The extremities may also be wrapped in flannels wrung out in hot water and covered with dry cloths. Some patients are greatly refreshed and helped by cool affusions, 70° to 80°, poured over the back and legs. In painful cases, relief can be obtained sometimes by applying the Charcot douche at a very high temperature to the back. The temperature at first is 90° F. This is gradually increased to about 160° F. The duration of treatment should be only about one minute.

Spermatorrhœa.—Cold sitz baths may be given for from five to twenty minutes, 50° to 70°, daily at bedtime.

Impotence.—Brief cold sitz baths, daily, at 56° to 64°, for from one to five minutes. The psychrophore, *i.e.*, application to the prostate of cold by a rubber condom or bladder secured over a rectal irrigator *au double courant*, is sometimes helpful.

Incontinence of Urine.—In paresis of sphincter or detrusor, brief cold sitz baths, daily, 56° to 64°, one to five minutes, are indicated; also cold rain baths (50° to 60°) and douches as general tonics. In spasmus detrusorum vesicæ, on the contrary, prolonged lukewarm sitz baths daily for thirty to sixty minutes, at 70° to 90°, should be given.

Electro-Therapeutics.

High and Low Potential Currents—Currents of "Tension" and "Quantity."—The current of the faradic battery varies in quality in accordance, 1, with the length and number of turns in the coil; 2, the form of the electrical wave; 3, the number of vibrations; and, 4, the strength of the battery.

The currents from short coils of coarse wire have a lower potential and slightly more voltage. They are more efficient in producing muscular contractions and are more irritating to the sensory nerves. These qualities are increased with slow interruptions of three or four per second. When muscular contractions and mechanical exercise with stimulation are desired the short coils (primary or secondary) with slow interruptions are indicated. The current from long coils has a higher potential and less voltage; it has less power in contracting muscles and a different effect on the sensory nerves. When the interruptions are very rapid and the coil is very long, the effect on the sensory nerves seems more sedative, perhaps in part because of a change in the form of the electrical waves.

Instrument makers have devised instruments with long coils (1,500 yards) which are tapped at three places, so that with one coil it is possible to get a current of low potential (or "quantity" as it has been called) or of high potential (or "current of tension"). The vibrator is made also so that currents of extremely rapid interruption can be obtained. These instruments are expensive, but are of special use to the neurologist in those cases in which he desires to apply faradism for spasm or neuralgia. They are said to be very useful in gynæcological work. Dr. Rockwell has advocated their use and shown their value. My own experience with the very high tension long coils is slight, but the theory on which they are based is sound; and the fact of their peculiar physical properties is attested by Mr. Kenelly.

The physiological effect of a current depends, as I have stated, in part upon the character of the wave of electrical force. If this is high and sharp, the stimulation is different from that produced by

a wave which gradually rises to its height. D'Arsonval has devised an instrument for producing these blunt-topped waves, and at the same time reversing the current. The current is called *sinusoidal* and is produced by revolving magnets, and the machine is expensive and complicated. The same result, I am told by Mr. Kenelly, is approximately produced by the faradic machine with long coils and very fine interruptions by means of a vibrating band, as described above. I am not aware of any special therapeutic results in neurology from this kind of current.

Vibratory Therapeutics.

Some relief of pain and spasm is secured by mechanical vibrations applied generally or locally. The most efficient means for this purpose is a vibrator with various terminals, which is run by hand, or by a motor. By means of a vibrating metal helmet applications can be made to the head, and by a vibrating chair the whole body can be shaken. This produces very rapid vibrations and a kind of benumbing of the part. The art of applying vibration in slower oscillations has been fully developed by Zander and is carried out in various Zander institutes, of which there are many in Europe and two in New York.

Suspension.

Suspension by the head and arms in a Sayre apparatus is occasionally used. The patient is raised from the ground for from one to three minutes, daily or triweekly. Or while seated in a chair he is partly lifted from his seat for from five to fifteen minutes. This measure will sometimes give relief in locomotor ataxia, paralysis agitans, sciatica, lumbago, and chronic myelitis. Its application requires care. According to Bogroff it produces a slight stretching of the cord and membranes and slight meningeal congestion.

Organic Extracts.

The Thyroid Gland.—This is used successfully in the treatment of myxœdema, certain forms of insanity, and in some cases of obesity. It does no good in other diseases so far as known. It is given usually in the form of tablets of the dried gland in doses of gr. v., gradually increased in some cases to gr. lx. daily.

Orchitic Extract.—There is some evidence that the extract of testicle has a tonic effect on the nerve centres.

The value of other organic extracts in nervous diseases is still undetermined.

The Rest Treatment.

This form of treatment was devised and perfected by Dr. S. Weir Mitchell and has been popularized by him and his pupils. Its value in many types of neurasthenia and hysteria, especially among women, is unquestioned. It seems more difficult to apply it to men, though it is often useful with them.

Among women it answers best in my experience for young women or those this side of middle life, of not very forceful character and not very strong will. The active, keen-witted, intellectual woman who suffers from headache, brain tire, and exhaustion from slight exertion does not do so well under a method which for a time renders the patient entirely abulic.

The essential features of the rest cure are: Isolation, diet, rest in bed, massage, electricity, and the energizing personality of a wise physician.

Isolation.—The patient can rarely be treated successfully at home, even if the relatives are not allowed to see her. The best place is a comfortable room in a boarding-house or private hospital. A private room in a general hospital may answer. A special nurse is necessary, and she should be young, neat, careful, sufficiently intelligent and tactful, but not overeducated or one who feels too keenly her social position. Decayed gentlewomen are sometimes very interesting characters, but they make poor nurses. I am afraid of nurses with an English or Irish accent. She should be a stranger to the patient, rather than one who has nursed the patient in previous illnesses. She should preferably know how to give massage, electricity, and the simpler forms of water treatment. The patient sees only the doctor and a *masseuse* if the nurse does not know how to give this treatment.

The diet should consist as far as possible of milk. During the first two weeks this is especially important. Skim-milk is used and it should be perfectly fresh. Four ounces are given at first every two hours. This amount is increased to two or four quarts a day. The milk may be treated in various ways in order to make it more palatable or digestible. The addition of a little salt or lime water, or of tea, coffee, or cocoa, or Vichy water, accomplishes this end. It may be varied with malted milk, dextrinized barley, Nestlé's food, or the milk may be mixed with barley or rice water. Two or four ounces of liquid malt may be given before the milk three times a day. At the end of a week a pint of beef tea is added. It is made (Mitchell) by chopping up one pound of raw beef and placing it in a bottle with one pint of water and five drops of strong

hydrochloric acid. This mixture stands all night, and in the morning the bottle is set in a pan of water at 110° F. and kept two hours at about this temperature. Strain through a stout cloth and squeeze the mass till nearly dry. The resulting fluid is given in three portions daily. If the taste be objected to, the meat may be roasted a trifle on one side, or the ordinary commercial extracts may be used. It is better at first for the patient to be fed by the nurse. The milk and other food should be taken slowly.

Rest.—It is extremely important that the patient be made to go to bed and lie flat on her back, not even sitting up to be fed. The object is not only to secure absolute rest, but to make the patient feel that she is in the hands of her physician, who is to manage her till she is well. The enforced quiet also adds to the desire later to to get well and regain her freedom. She is to be kept in bed for a month; then allowed to sit up for ten minutes twice a day, this time being lengthened by five or ten minutes daily. After five or six weeks she may be allowed a drive or a short walk. About this time one must also begin systematically to enforce walking and other exercise, and the patient must be taught to ignore the slight pain and fatigue that at first ensue.

Massage.—The system of massage recommended by Mitchell is simpler than that ordinarily used by the Swedish professionals, and consists mainly of kneading and centripetal friction. It is given at least an hour after a meal and lasts at first fifteen or twenty minutes once a day. In a few days this is increased to three-quarters of an hour and an hour. The legs are *masséd* first, then the abdomen, chest, and arms. The head and neck are not touched. No ointment or lubricating substance is used. Particular attention is paid to the abdomen in order to prevent constipation. After massage there should be a rest of an hour. After four or eight weeks Swedish movements are added to the massage.

Electricity.—The faradic current is used with a primary coil and a slow interrupter. In very sensitive persons a long secondary coil with fine interruptions is better. Electrodes of the "normal" size are employed and each segment of the limbs is gone over, beginning with the periphery. The two electrodes are placed over the muscles a few inches apart and each muscle is contracted four or six times. The question of poles may be ignored. The feet, legs, abdomen, back, and arms are gone over successively, then the fine secondary current is turned on. A large electrode, of the "indifferent" size, is placed on the back of the neck, and the other electrode placed on the sole of one foot; a current is passed for seven and a half minutes; then the electrode is shifted to the other foot and the

current given for the same time. It is best for the physician himself to give the electricity if possible. The duration of each séance is from three-quarters to one hour, and it is continued for six weeks. Very mild currents are used at first.

The duration of the rest-cure should never be less than a month. It is usually about six or eight weeks. A typical schedule for a rest-cure patient as given by Dr. John K. Mitchell is the following:

7 A.M.—Cocoa.
Cool sponge bath with rough rub and toilet for the day.
8 A.M.—Milk, breakfast.
Rest an hour after.
10 A.M.—8 oz. peptonized milk.
11 A.M.—Massage.
12 M.—Milk or soup.
Reading aloud by nurse
1:30 P.M.—Dinner.
Rest an hour.
3:30 P.M.—8 oz. peptonized milk.
4 P.M.—Electricity.
6 P.M.—Supper with milk.
8 P.M.—Reading aloud by nurse one-half hour.
9 P.M.—Light rubbing by nurse with drip sheet.
8 oz. malt extract with meals, tonic after meals.
8 oz. peptonized milk with biscuit at bedtime and a glass of milk during the night if desired.

Laxative: cascara, 10 to 30 drops p. r. n. Later Swedish movements are added to the massage.

Additional Measures.—A sponge bath is given every morning. Insomnia is to be feared at the start, and for this bromide of sodium may be given in doses of gr. xxx. at 6 and 9 P.M. and gradually decreased grain by grain; or sulphonal (gr. xx. to xxx.) in hot water, or trional (gr. xv.). Some form of hydrotherapy may answer better than drugs, and a favorite measure is the drip sheet.

The following are the directions for its use (Mitchell):

Basin of water at 65° F. Lower the temperature day by day by degrees to 55° F., or to still less. Put in the basin a sheet, letting the corners hang out to be taken hold of. The patient stands in one garment in comfortably hot water. Have ready a large soft towel and iced water. Dip the towel in this, wring it, and put it turban-wise about the head and back of the neck. Take off night-dress. Standing in front of patient, the basin and sheet behind, the maid seizes the wet sheet by two corners and throws it around

the patient, who holds it at the neck. A rough, smart, rapid rub from the outside applies the sheet everywhere. This takes but two minutes or less. Drop the sheet, let the patient lie down on a lounge upon a blanket, wrap her in it, dry thoroughly and roughly with coarse towels placed at hand. Wrap in a dry blanket. Remove ice wrap; dry hair; put on nightdress. Bed, the feet covered with a flannel wrap.

As tonics, lactate, pyrophosphate, or subcarbonate of iron are given in doses of gr. xxx. daily. Small doses of strychnine, salicin or quinine, or dilute phosphoric acid may be useful.

The partial rest-cure is indicated in the milder cases of neurasthenia and hysteria. The following is the schedule given by Dr. Weir Mitchell:

A.M.—On awaking, cup of cocoa. Take bath. (Temperature given.) Lie down on lounge while using drying-towels; or, better, be sponged and dried by an attendant. In this process the surface to be rubbed red, or, if drying one's self, to use flesh brush. Bed or lounge again. Breakfast. Before each meal take three ounces of malt extract; aperient at need in malt. Tonic after each meal. Detail as to breakfast diet. If eyes are good, may then read seated in bed. At 10 to 11 A.M., one hour's massage. Rest one hour; may be read to, or read if eyes are good, or knit. At this time, 11 A.M., four ounces of beef soup or eight ounces of milk. At noon may rise, dress slowly, resting once or twice a few minutes while dressing, and remain up until 3 P.M. See children, attend to household business; see one visitor, if desirable. From 1 to 1:30 P.M. malt, etc., and lunch. Detail as to diet. At first, as a rule, let this meal represent dinner. Tonic, and after it to rest on a lounge, occupied as above, reading or being read to. If possible, drive out or use tramway, so as to get air. Walk as little as possible. On return from drive repeat milk or soup. About 5 P.M., electricity, if used at all. Rest until 7 P.M. Supper at 7 P.M. Detail as to meal. Malt as before, with or without aperient, as occasion demands. Tonic. To spend evening with family as usual. Best not to use eyes at night for near view. Bed at 10 P.M. No letters to be written for two months, when most of these details have to be revised.

After two months of massage it should, in these cases, as in complete rest, be used on alternate days, and by degrees given up. If the nurse or *masseuse* is able to teach the patient the use of Swedish movements, it is desirable that these or some definite slowly increased system of chamber gymnastics be continued for months. Finally, walking must be resumed with slow and system-

atic increase. After the second month write out a schedule of less restriction, to be followed for six months.

SPECIAL THERAPEUTICS.

ALCOHOLISM.—

℞ Strychninæ nitrat., ... gr. i.
Aquæ destillat., ... ʒ v.
Acidi carbolici, ... gr. i.
M. Sig. ♏x. hypodermically q. 4 h.

℞ Atropinæ sulph., ... gr. ¼.
Strychninæ nitrat., ... gr. i¼.
Glonoini, ... gr. ½.
Tinct. strophanth., ... ʒ iij.
Extr. cinchon. fl., ... q.s. ad ℥ vi.
M. Sig. ʒ i. t.i.d.

ACUTE ALCOHOLISM.—

℞ Sodii bromid., ... ℥ i.
Tinct. capsici, ... ʒ ss.
Chloral. hydrat., ... ℥ ss.
Aquæ menth. pip., ... ℥ iij.
M. Sig. ʒ i. q. 4 h.

EPILEPSY.—A method of treating epilepsy has been recommended by Dr. P. Flechsig as being successful in some obstinate cases. It consists in giving opium in doses of gr. ½ t.i.d., increased to gr. iij. or v. t.i.d. This is kept up for five or six weeks. The patient is then placed at once on large doses of bromide of potassium. I have found in applying this treatment that a short course of opium enhances the subsequent effect of the bromide. It is not always wise or possible to give large doses of opium for so long a time as six weeks.

I have insisted upon the necessity of giving large doses of bromides for a time in all cases of epilepsy. Dr. Ch. Féré recommends regularly a gradual increase of bromides from ʒ i. daily to ʒ iv. or ʒ v. Such doses diminish attacks from one or two weekly to one monthly. The criteria to be depended upon in advising such treatment are the weight of the patient and the number of attacks. If the weight falls and gastric trouble ensues, or if the attacks are not lessened, the drug should not be increased.

In many cases it is not wise to continue the alkaline bromides indefinitely, on account of the condition of the bladder and urine. The latter becomes cloudy, feebly acid or alkaline, and the bladder is irritable. The following prescription I find most useful as an adjunct to the ordinary formulæ of bromides:

℞ Acidi hydrobromici dil., ... ♏ x. to xx.
Ferri bromid., ... gr. i.
Potas. bromid., ... gr. v. to x.
Sodii salicylat., ... gr. ij.
Spts. rectificat., ... ♏ x.
Glycerol. pepsin., ... ♏ x.
Olei gaulth., ... ♏ ½.
Liq. ammon. citrat. (Br. P.), ... ♏ xxx.
M. Sig. i. dose. This may be doubled or tripled.

Antifebrin and *sulphonal* are drugs which sometimes control the convulsions. Antifebrin may be given in the form of a five-grain tablet *ter in die* with a bromide solution. Sulphonal is given best in the form of a powder at night. Fifteen to twenty grains can be administered then. I do not think the drug a very useful one. Trional in fifteeen-grain doses is better.

The following are some special prescriptions which may be used in epilepsy:

℞ Antipyrin, ... ʒ iss.
Ammon. bromid., ... ℈ xvi.
Aquæ, ... ʒ iv.
M. Sig. ʒ ij. t.i.d.
To be used without other medication except tonics.

℞ Beta-naphthol, ... ʒ i.
Bismuth. salicylat., ... ʒ ss.
M. Sig. i. daily in two doses with ʒ i. to ʒ v. potas. bromide daily or with gr. xlv. borax.

℞ Ext. belladonn., ... gr. xv.
Stannii oxid., ... ʒ i.
M. Div. in pil. No. lx. Sig. i. A.M. and P.M.
To be used with bromides.

℞ Trional, ... gr. xv.
Sodii bromid., ... gr. xx.
M. Sig. i. dose.
At night in nocturnal epilepsy.

Pure bromine combined with cod-liver oil or sesame oil can be given in doses of gr. x. to gr. xxx., and sometimes acts well. Removal of salt from the diet is thought important by some.

The Surgical Treatment of Epilepsy.—This is indicated only in traumatic forms of comparatively recent character, before more than twenty-five fits have occurred. It is much more indicated in local or Jacksonian epilepsy.

The mortality from the operation is seven per cent. Among 159 cases collected by Laurent and Agnew there were 58 cured, 52 improved, 28 unimproved, 11 died. Among 42 cases collected by

Starr ("Brain Surgery") there were 13 cured, 11 improved, 15 not improved, 3 died. The term "improved" has little meaning.

HYSTERIA.—

℞ Zinci valerianat., ... ʒ iij.
Ext. sumbul., ... ʒ ss.
Quininæ sulph., ... ʒ i.
M. Div. in capsulæ No. lx. Sig. i. t.i.d.

℞ Spts. ammon. aromat.,
Spts. lavandul. co.,
Spts. ether. co., ... āā ʒ i.
M. Sig. ʒ i. p.r.n.

Apomorphine $\frac{1}{8}$ gr. hypodermatically and pilocarpine $\frac{1}{10}$ gr. in the same way often break up a hysterical crisis.

IMPOTENCE.—In the treatment of impotence by electricity the galvanic or faradic current may be used. My own method is to combine the two, using the De Watteville switch. The positive pole with a large electrode is placed over the dorso-lumbar region, the negative pole with normal or large electrodes is placed on the perineum. A current of from 5 to 10 milliamperes is turned on. Then the faradic current is added. The secondary long coil with fine vibration may be employed if there is much irritability; the short coil with coarse vibrations if there is anæsthesia or great insensibility. The current is passed with occasional interruptions for five minutes. The lower electrode is then moved for a time to the root of the penis and the current passed for a minute. A steel sound as large as the urethra will hold is then introduced and the lower (negative) electrode connected with it. The galvanic current is reduced to 2 or 3 milliamperes, the faradic made as strong as the patient can stand it. The application lasts two or three minutes.

In addition I employ hypodermic injections of strychnine, or cantharidate of potassium (gr. $\frac{1}{200}$). Cupping and ligature of the dorsal veins have been successfully used.

NEW DRUGS FOR INSOMNIA.—There are no good medicines for insomnia, but the following may be occasionally used:

Amyl Hydrate.—This is a fairly good and safe hypnotic, but disagreeable to the taste. The dose is about a drachm, given in syrup and water. It has a pungent taste like paraldehyde and sometimes disturbs the stomach.

Chloral-amide is a good hypnotic, acting like chloral hydrate, but more slowly. It is less irritating and is safer than chloral,

though the dangers of the latter drug are very slight. The dose is 15 to 45 grains in powder or dissolved in alcohol or tincture of cardamom.

Duboisine sulphate has been warmly recommended as a hypnotic in doses of gr. $\frac{1}{80}$ to gr. $\frac{1}{50}$. It has about the same effect as hyoscine.

Trional is a disulphone, or diethylsulphon-methylethylmethan. It is closely related chemically and therapeutically to sulphonal. It is a white powder slightly soluble in water and best given in milk or wine. It acts in from ten to thirty minutes and produces a quiet sleep. It is an excellent hypnotic, ranking with sulphonal and having the advantage of acting more promptly. The dose is from 10 to 40 grains. At least 30 grains are needed in bad cases.

Locomotor Ataxia.—Erb has recently reported many cases showing that the inunction of mercury is followed by decided benefit in this disease. He accompanies or follows the "cure" with electricity, nitrate of silver, baths, and tonics.

Leyden has strongly advocated what may be called the tonic, expectant, and training treatment of tabes. He does not expect much of drugs, but depends on diet, baths, exercise, and quiet. This is very well so far as it goes, but is a most unfortunate view as a whole, for it takes no account of the fact that in tabes there is a specific something continually at work eating away the spinal cord. The physician must find an agent to counteract this. Rest and proper nourishment and exercise do it to some extent.

The Bonuzzi method of stretching the spinal cord is advocated by Benedict. It is only a modification of subcutaneous stretching and was practised in my clinic ten years ago. The patient lies upon the back, the head maintained in an elevated position by a bolster; the lower extremities are then flexed upon the body, forming a semicircle, the knees being placed upon the chest of the patient and the legs held straight; the operator seizing the diverging ankles carries them strongly toward the floor. This apparently difficult manœuvre is in reality easily executed.

The Exercise Treatment of Locomotor Ataxia.—The treatment of locomotor ataxia by means of systematic exercises for training the ataxic limbs often produces some very satisfactory results. The method was elaborated first by Dr. Fränkel, and still further by Dr. Hirschberg. For the convenience of students and readers, a schedule of the exercises which I prescribe, and which are based, more or less, upon those of the authors mentioned, is appended here.

The exercises are usually to be taken twice a day, and each exercise is to be done with the utmost care and precision by the patient.

Exercises for the Hands and Arms.—1. Sit in front of a table, place the hand upon it, then elevate each finger as far as possible. Then, raising the hand slightly, extend and then flex each finger and thumb as far as possible. Do this first with the right and then with the left. Repeat once.

2. With the hand extended on the table, abduct the thumb and then each finger separately, as far as possible. Repeat three times.

3. Touch with the end of the thumb each finger tip separately and exactly. Then touch the middle of each phalanx of each of the four fingers with the tip of the thumb. Repeat three times.

4. Place the hand in the position of piano playing and elevate the thumb and fingers in succession, bringing them down again, as in striking the notes of the piano. Do this twenty times with the right hand, and same with the left.

5. Sit at a table with a large sheet of paper and pencil, make four dots in the four corners of the paper and one in the centre. Draw lines from corner dots to centre dot with right hand; same with left.

6. Draw another set of lines parallel to the first, with the right hand; same with left.

7. Throw ten pennies upon the paper, pick them up and place them in a single pile with the right hand; then with the left; repeat twice.

8. Spread the pennies about on the table, touch each one slowly and exactly with the forefinger of right hand; then with forefinger of left.

9. Place an ordinary solitaire board on the table, with the marbles in the groove around the holes. Put the marbles in their places with right hand; same with left hand. Patient may, with advantage, practise the game for the purpose of steadying his hands.

10. Take ordinary fox-and-geese board with holes and pegs, and, beginning at one corner, place the pegs in the holes, one after the other, using first the right hand, then the left.

These exercises should be gone through with twice a day, and should be done slowly and carefully, with a conscious effort every time of trying to do one's best.

Exercises for the Body and Lower Limbs.—1. Sit in a chair, rise slowly to erect position, without help from cane or arms of chair. Sit down slowly in the same way. Repeat once.

2. Stand with cane, feet together, advance left foot and return it. Same with right. Repeat three times.

3. Walk ten steps with cane, slowly. Walk backward five steps with cane, slowly.

4. Stand without cane, feet a little spread, hands on hips. In this position flex the knees, and stoop slowly down as far as possible, rise slowly; repeat twice.

5. Stand erect, carry left foot behind, and bring it back to its place; the same with the right. Repeat three times.

6. Walk twenty steps, as in exercise No. 3; then walk backward five steps.

7. Repeat exercise No. 2, without cane.

8. Stand without cane, heels together, hands on hips. Stand in this way until you can count twenty. Increase the duration each day by five, until you can stand in this way while one hundred is being counted.

9. Stand without cane, feet spread apart; raise the arms up from the sides until they meet above the head. Repeat this three times. With the arms raised above the head, carry them forward and downward, bending with the body until the tips of the fingers come as near the floor as they can be safely carried.

10. Stand without cane, feet spread apart, hands on hips; flex the trunk forward, then to the left, then backward, then to the right, making a circle with the head. Repeat this three times.

11. Do exercise No. 9 with heels together.

12. Do exercise No. 10 with heels together.

13. Walk along a fixed line, such as a seam on the carpet, with cane, placing the feet carefully on the line each time. Walk a distance of at least fifteen feet. Repeat this twice.

14. Do the same without cane.

15. Stand erect with cane; describe a circle on the floor with the toe of right foot. Same with toe of left. Repeat twice.

Between the fifth and sixth exercises the patient should rest for a few moments.

Masturbation.—An efficient remedy against this practice is to insert a piece of silver or copper wire through a portion of the foreskin at the edge of the glans. This is a rather heroic measure and called for more especially in those suffering from mental deterioration or insanity. A somewhat less severe measure is to paint the glans with cantharidal collodion.

Lumbar Puncture of the Spinal Cord.—Paracentesis of the spinal dura mater has been recommended by Quincke and Ziemssen for the treatment of meningitis with serous effusion. The relief obtained is usually but temporary, but the measure may be of help in diagnosis. I have tried it in a few cases with negative results, but have found that the operation is not difficult or dangerous if carefully done.

Quincke's directions concerning the operation should be followed —that is, the patient should lie on his left side with his lumbar spine flexed well forward; the needle is cautiously inserted to a depth of 5 cm. between the arches of the third and fourth or fourth and fifth lumbar vertebræ near the spinous processes.

Multiple Neuritis.—The pains and sensitiveness of the early stage may be met with powders of the following:

℞ Salophen, gr. xv., 4 in die;

or

℞ Sodii iodid.,
Sodii salicylat.,
Antipyrin, ... āā gr. v.
M. Sig. i. three to four times daily.

Salicylate of potassium or sodium in large doses sometimes helps the pain, but as a rule it does no good. Croton chloral in doses of gr. v. may be used for a time. Later one should give ferric iodide and potassium iodide in small doses every four hours, alternating with phosphorus (gr. $\frac{1}{60}$). Still later, that is to say, five or six weeks after full development of the disease, give hypodermic injections of strychnine. If the patient is very alcoholic I give strychnine in the first week. Static sparks, galvanism, or faradism should also be administered in brief daily séances for a period of six weeks. Galvanism is best used first, later fine high potential faradic currents or sparks.

Drugs for Neuralgia, Headache, Painful Affections.—So far as I have been able to discover, none of the new drugs surpass in value antipyrin, antifebrin, and phenacetin, or chloral, chloralamid, paraldehyde, and sulphonal. Some of them are, however, useful in combination or alternation with the older preparations; some are more palatable, convenient, or may cause fewer unpleasant symptoms. It is to be supposed that most of the new drugs have some commercial house behind them which is interested in popularizing their use. This has to be considered in estimating the value of some published reports.

External Applications for Pain.—

℞ Spts. chloroformi, ... ʒ ij.
Alcohol, ... ℥ iij.
Menthol, ... ʒ vi.
M. Sig. Ext. use.
Paint on part and cover with bandage.

Sciatica.—In chronic and in early cases the rest treatment as indicated in the body of my book will usually be of most service.

The leg should be carefully and firmly enveloped in a flannel bandage from toe to hip. Then a Thomas splint is applied. Every day the splint is removed and the leg cautiously exercised. At the end of two or three weeks the patient can be allowed to use the limb himself for a time. With the flannel bandage, an ice bag or hot-water bag is not always needed.

The experience of the clinic at La Salpêtrière is that suspension does good service in sciatica.

Tic Douloureux.—Local injections of five or ten drops of four-per-cent or two-per-cent solution of cocaine often give relief for a considerable time. The chloride of methyl spray or a bit of cotton wet with the methyl and drawn over the affected area until it is frozen will prove helpful. I have also found the Granville *percuteur* gently applied for five or ten minutes of service. Along with such measures one should give pills of aconitine gr. $\frac{1}{200}$ every four hours and a tonic as follows:

℞ Acidi phosphor. dil., ... ℥ i.
Ferri pyrophosphat., ... ʒ i.
Quininæ sulph., ... ʒ i. to ʒ ij.
Aquæ, ... ℥ iij.
M. Sig. ʒ i. t.i.d.

Croton chloral in doses of five to fifteen grains three times a day may be substituted for the aconitine, and codeine or cannabis indica combined with it.

In cases of not too long standing, rest and the hypodermic injection of strychnine, as described in the body of this work, should always be prescribed.

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